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ABSTRACT

Theories regarding organic determinants of learning and behavior disorders are reviewed historically. Cases illustrating how a bio-ecologic examination can isolate the substances to which a person reacts and some of the reasons for those reactions are presented; and the role of various disorders in relation to the central nervous system is discussed. From ancient times, it has been observed that reactions do occur to environmental substances. Case studies of five children (2 to 12 years old) demonstrate that reactions to environmental substances do occur, and that these reactions are observed to be metabolic disorders stemming largely from a state of addiction (sometimes with genetic predispositions) rather than stemming from allergies. Discussions cover the role of immunologic and nonimmunologic inflamatory reactions, adaptive addiction, endorphins, disordered amino acid metabolism, approach and avoidance, tranquilizers, and anticonvulsants. The appendix, which makes up the bulk of the document, details the five case histories. Presented within each case study are sections on the initial examination and history, methods of laboratory examination, results of laboratory examination, formulation, treatment, prognosis, and diagnoses (including listings of foods causing allergic reactions). Among formulations from the cases are the following: a cause and effect relationship between central nervous system malfunction and environmental substances both in foods and chemicals was established in a 6-year-old girl; specific goods evoked a wide assortment of central nervous system symptoms in a 7-year-old girl, including grimacing, tension, irritability, shaking, and hyperactivity; and a 12-year-old boy was found to have depressive and hyperactive reactions to several foods. (SW)

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ORGANIC DETERMINANTS OF LEARNING AND BEHAVIORAL DISORDERS

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INTRODUCTION

The axiom, "One man's meat is another man's poison", has been with us since ancient times. Hippocrates, the Father of Medicine, observed that if a commonly used food was avoided for as much as four days, sometimes symptoms emerged on reexposure to that food. In England in 1621, R. Burton published the book, The Anatomy of Melancholy in which he observed cow's milk to be a frequent cause of depression and headache. In 1843, Charles Dickens penned his famous, A Christman Carol, in which Jacob Morley, Scrooge's former partner, appeared to Scrooge in a ghostly apparition, "You don't believe in me", observed the ghost. "I don't", said Scrooge. "What evidence would you have of my reality beyond that of your senses?" "I don't know", said Scrooge. "Why do you doubt your senses?" "Because", said Scrooge, a little thing effects them. A slight disorder of the stomach makes them cheat. You might be an undigested bit of beef, a blot of mustard, a crumb of cheese, a fragment of undone potato. There is more of gravy than the grave about you, whatever you are!"

In 1973, F.C. Dohan published objective controlled experimental evidence that schizophrenics were made worse by chronic exposure to wheat gluten. In 1974, I (W.H. Philpott) illustrated to Dr. Dohan a wheat reaction in a mental patient. Within fifteen minutes of eating a provocative test meal of wheat, she stood up saying the room was very hot. A moment later, she fell to the floor, limp and unconscious. Intravenously, I gave her vitamin C, B_6, calcium and magnesium with which she promptly awakened and was symptom free. Dr. Dohan observed there will be very few psychiatrists that will be capable of believing that this was a food reaction. They will be sure that it is something that we were talking about that evoked her symptoms. This resistance to believe that a food or chemical can evoke a central nervous system disorder ranging from hyperkinesis, learning disorders and behavioral disorders to psychosis depicts tremendous resistance. Through the strongly entrenched educational system, modern psychiatrists, psychologists and educators by and large hold strongly to the belief system that interpersonal relationships are the central causative factor of learning disorders, behavioral disorders and even psychosis. This is put forth as a hypothesis but clinically treated as fact in which the patient through deductive reasoning is fit in to the belief system of the professional in which anger, disturbed interpersonal relationships and so forth are taken as evidence of primary cause of the condition rather than viewed as the secondary conditions produced by the organic state evoked by reactions to environmental substances, inherent metabolic disorders, associated nutritional deficiencies and so forth. A communication conflict exists between those psychiatrists,

psychologists and educators who claim they have objective evidence that reactions to environmental substances are only placebo effect and those who equally claim objective observations relative to the evidence of maladaptive reaction to environmental substances evoking a wide assortment of central nervous system disorders including learning and behavioral disorders.

There is a logical resistance to believing in the reality of these maladaptive reactions to environmental substances due to the training psychiatrists, psychologists and special educators receive. I was as resistant as anyone else in accepting this evidence, and even though I had heard of this in my senior year of medical school and again during psychiatric residency training, I always discounted it. It wasn't until some twenty years after I had originally heard of this possibility that I was a party to a double-blind study which gave evidence of a statistical value that I dared to have a belief in the possibility of environmental factors evoking central nervous system disorders. There have been a number of single-blind, double-blind and otherwise objective examinations confirming the evidence that environmental factors do enter into evoking varied degrees of central nervous system disorders. At the same time, there are those who have performed faulted experiments in which the basic principles governing these reactions have been ignored and or even purposely left out in which the conclusion was that these reactions to environmental substances are only placebo effects. These experiments have been faulted by ignoring and or even denying the existance of the adapted state of addiction to foods and chemicals, and therefore these experiments did not arrange for an initial avoidance period in which there would be a chemical restitution from addiction before provocative testing began. The other area of faulting of these experiments has been that of looking for a single symptom such as an engorgement of the mucous membrane of the nose and ignoring any other symptoms when it is well-known that reactions to environmental substances can react in any part of the body, not just the nose.

The central issues in these disputes have centered around those who maintain an immunologic model and expect these maladaptive reactions to behave the same as an IgE mediated response and those who have found that very seldom do these or other immunologic responses bear any relationship to these maladaptive responses, but instead they can be monitored in the laboratory and demonstrated to have an assortment of disordered metabolic functions such as disordered acid-base balance, disordered and or genetic amino acid disorders, carbohydrate disorders, toxic states and so forth.

Another factor that is frequently ignored is that central nervous system malfunction and or frank organic disorders such as may be produced by birth trauma, infection, traumatic

injury and so forth may so disorder the chemistry of the body and particularly the brain as to predispose to maladaptive reactions to environmental substances. Classically, a neurological examination determines evidence of organicity but does not determine evidence of the degree of reversibility of that organic state. It is by and large assumed that little or no reversibility exists, simply because it is known that the central nervous system is not self-repairing, and often it is not appreciably considered that an improved biochemical homeostatic state of the patient may markedly improve the function of an organic state. A bio-ecologic examination determines not only organicity but degree of functional reversibility of that organicity. We professionals (neurologists, educators, psychologists and psychiatrists) are something like the three blind men who examined an elephant in which one examined the trunk, another the leg and another the tail. From their descriptions, it could not be determined that they all had a common source, the elephant. A bio-ecologic examination has provided us the overview of what we are dealing with. In this, provocative testing objectively observed has been used to observe the reaction that is produced on exposure, and at the same time the disordered chemistry is monitored judging the difference between a nonreactive state and a reactive state. This system of examination clarifies the issues and provides a workable clinical format for progress in dealing with these disorders. The evidence of disordered chemistry emerging during such provocative testing gives more convincing evidence than double-blind studies based on evoked symptoms can provide. The relief of symptoms obtained by honoring these findings provides even more convincing evidence as to the fact that such an examination is indeed on course.

There is considerable more knowledge in medical science than is being applied clinically. It is estimated that it often takes twenty years for a scientific established fact to become routinely and profitably applied in medical practice. There is considerably more reliable information in the area of clinical ecology and nutrition than there are professionals skilled to render these necessary services. The more highly skilled an educated person becomes, the less likely he is to change due to the strongly learned conditioning process. It has been observed that physicians start out as medical students with more than average obsessive-compulsiveness, and the demands of exactness of their medical education reinforces this obsessive-compulsiveness. The reason this is so important is that obsessive-compulsiveness mediates against the ability to change. The physician feels comfortable when following the orientations he has learned in medical school and residency training and feels anxious about altering these orientations. The public is not so strongly conditioned in their behavior and formulations as professionals and can change more readily when new and useful information emerges. The public's request for specific medical and educational services gradually helps professionals change.

Also, we have to consider that professionals are doing more than using scientific information. They are equally rendering a consumer service, and they are unable to earn a living performing a specific service until there is sufficient public demand for that service.

CASE HISTORIES

See Appendix for details of Case Histories 2, 3 and 4.

Case History #1

Charlie is a seven year old white girl with multiple grand mal seizures since age two. She had five grand mal seizures during the initial hour of examination. There have been multiple neurological examinations and trials on multiple medications. She was receiving three medications at the time of initial examination. She did not talk, showed no evidence of learning, was chronically dizzy and had to be led by the hand when walking. There was no indication that medication had ever reduced the frequency or intensity of her seizures. The mother had observed that the intensity and frequency of seizures were increased after some meals. This was reported to several physicians but always discounted as unlikely. Provocative testing established a cause and effect relationship between foods, chemicals and grand mal seizures and lapse states. A bio-ecologic treatment of avoiding symptom evoking foods and chemicals plus laboratory indicated supplemental nutrients including B_6 reduced the lapse states and seizures to a single grand mal seizure on awakening in the morning. Under these circumstances, anti-seizure medication was withdrawn without any increase in seizures. When the amino acid quantitative analysis was returned, it indicated that she was deficient in cystine. When cystine was added to her program, her seizures ceased within one week, and there has been no return of seizures during the last two years which includes even chicken pox with a fever.

Her motor function is normal. Affect is normal. She readily talks to her mother and asks the usual questions a child would ask. Her unfortunate experience has left her with a phobia of talking in public which requires behavioral modification.

This case illustrates how a bio-ecologic examination can be sufficiently specific to isolate by provocative test means not only the substances to which the person reacts, but some of the reasons for those reactions. Deficient cystine which

was discovered by an examination of her blood levels of cystine was demonstrated to be a specific key to the cause of her seizures that when it was supplemented, her seizures ceased. Cystine is necessary for its contribution of sulfur to B-complex vitamins without which they function poorly. Cystine is a precursor to taurine which is a necessary brain synaptic junction modulator of responses. It has been demonstrated that focal seizure areas in the brain are low in taurine, and that taurine does have anti-seizure value. There are many other reasons for seizures such as the inability to process carnosine, hyperammonemia, disordered or deficient calcium metabolism, magnesium deficiency, manganese deficiency, zinc deficiency, lead or mercury toxicity and so forth, all of which are considered in a bio-ecologic examination and in individual cases turn out to be the specific culprit.

Case History #2

Kerri is a seven year old white girl diagnosed by a neurologist as having Tourette's syndrome based on the symptoms of grimacing, rhythmic mouth movements associated with rhythmic flexion of the arms and clenching of the fists and generalized rigidity. She had poor motor coordination and reversal of sequences. She was attending a special education class due to her learning disorder. She had been diagnosed as being hyperactive. Provocative testing revealed an array of symptoms to emerge on test exposure to specific foods. These symptoms included reversal of sequence, wavy lines, inconsistent size of letters when writing the alphabet, grimacing, arm flexing, fist clenching, flat affect, social withdrawal and lack of response to environmental stimuli. Avoidance of symptom incriminated foods plus providing supplementation of specific laboratory indicated needs produced a symptom-free state. Within a two year period, her scholastic achievements were such as to place her up with her age group and in the middle of a traditional educational class.

This case reveals the very solid values often achieved by a bio-ecologic examination and treatment. It also reveals how significant it can be to take the extra step of a bio-ecologic examination and treatment beyond that of a traditional physical, neurological, psychological, educational diagnosis and treatment.

Case History #3

Michael is a seven year old white boy who had been diagnosed as having defective motor skills, dyslexia and hyperactivity as well as some degree of cerebral atrophy. Provocative

testing established a cause and effect relationship between his symptoms and maladaptive reactions to commonly used foods and chemicals. A test for wheat gave evidence of celiac disease. It was subsequently discovered that when he would eat any cereal grain containing gluten, he would become excessively hyperactive. Dairy products would produce the same type of hyperactivity and social disorder. One year later, a test for wheat did not produce the drop in alkaline phosphatase which brings in question whether this was celiac disease or simply a maladaptively active gastrointestional state at the time of initial examination and was a pseudo-type celiac disease. However, his symptoms of hyperactivity and social disruptive disorder emerged on the wheat test. It still is very important that he leave cereal grains out of his diet whether or not he has celiac disease. It still is necessary for him to attend a special school, however his behavior is markedly improved with the exception of a few times when he violated his program, especially obtaining cereal grains and or dairy products. On one of these occasions, he changed from his usual, calm and reasonable social behavior to hyperactive, aggressive and knocked a tooth out of a fellow classmate.

The CAT scan would suggest that there is an underlying organicity in this case. It is important to understand that organic deficits of the central nervous system, liver, pancreas or small intestine mucous membrane would predispose to reactions to environmental substances. Therefore, these cases should not be lightly dismissed as organic but should be given the opportunity for all possible symptomatic reversal through assessing their organic defects and the predisposition these defects make to reactions to environmental substances. Honoring their reactions to environmental substances and demonstrated nutritional needs can frequently materially reduce symptoms.

Case History #4

Roderick was two years old at the time of his initial examination. He had been hyperactive since birth as well as having chronic diarrhea and intestinal bleeding. Extensive medical diagnosis and treatment had not solved his problem. A bio-ecologic examination using provocative testing and laboratory assessment of nutritional needs and metabolic disorders revealed a cause and effect relationship between his hyperactivity and specific foods, as well as the entire gastrointestinal tract maladaptively reacting to an assortment of foods. He was demonstrated to have gastritis, celiac disease, Crohn's disease, colitis, lactase deficiency, a chemical stage of diabetes mellitus, mineral deficiencies and amino acid deficiencies. When placed on the appropriate program of avoidance and spacing of contact with reactive foods and supplementing nutritional needs, he has become

symptom free and has remained so for the last seven months other than when his program was violated. On one occasion, he ate some cracker crumbs and promptly had diarrhea.

Case History #5

Steve is a twelve year old white boy. As an infant, he was hyperactive, had a violent temper, frequently crying, rocking, screaming and never satisfied. Milk was not tolerated. There were frequent upper respiratory infections during infancy and childhood. He is hostile, demanding, throwing things and dependent. Attention was poor, memory difficult and confused. Concepts were fragmented. He had limited language development and poor visual motor integration. He had been treated with central nervous system excitants including dextroamphetamines and Cylert. Provocative testing established an obvious cause and effect relationship between foods and his symptoms. Deficits were found in vitamins, amino acids and minerals. Bio-ecologic treatment achieved success more than any other treatment given.

DISCUSSION

The Role of Immunologic and Nonimmunologic Inflammatory Reactions

Although it is observed that immunologic (allergic) reactions can evoke central nervous system disorders, it is our observation that only a small percentage of observed central nervous system reactions are thus evoked. The majority of central nervous system reactions evoking hyperkinesis, learning disorders, autism, mental illness and seizures are observed to be an assortment of metabolic disorders which are nonimmunologic (nonallergic). The majority of physicians have used the immunologic model by which they have judged the reality of these reactions disordering central nervous system function. Since these could not be demonstrated to be immunologic in origin, it engendered doubts as to the existence of these reactions, and therefore they were often assumed to be placebo effects resulting from the anticipation anxiety of the patient or the excessive enthusiasm of a nonobjective physician. Biochemical monitoring has revealed an assortment of metabolic disorders associated with objectively observed symptoms emerging during provocative testing. By this means, the reality of the responses have been documented but also to some degree the disordered chemistry causing these reactions. It should be understood that there is no better way to develop an anticipation anxiety which can create a placebo effect than to have bona fide organic reactions which are then extended by these anticipatory

responses, so therefore the symptoms observed in a patient are always a combination of bona fide organic reactions and anticipation reactions.

The Role of Adaptive Addiction

Biological addiction can be described as a state of biological adaptation in which there is relief on contact with the addictive substance and symptoms on avoidance of the addictive substance. Most reactions to foods and chemicals can be demonstrated to be of the nature of an adaptive addiction. With five days of avoidance, usually there is a biological recovery from the state of addiction, after which exposure to the substance will produce an acute reaction within a few minutes rather than a delayed withdrawal symptom reaction. Adaptive addiction is a chronic state of stress leading to biological degeneration, and thus degenerative diseases, either physical or mental. Addiction is observed to disturb acid-base balance, carbohydrate metabolism, lipid metabolism and amino acid metabolism. This can be demonstrated by monitoring during provocative testing. These acute test reactions are the miniature of degenerative diseases. When adaptive addiction can no longer be maintained, there emerges maturity-onset diabetes mellitus with its large assortment of associated complications. Prior to this chronic state of disordered carbohydrate metabolism named as maturity-onset diabetes mellitus, varying degrees of these complications are often named as specific diseases simply because of the fact a major organ system is involved in symptom production.

Psychological addiction is a psychological elaboration of biological addiction and is based on the pain-pleasure principle. In biological addiction, relief of discomfort is achieved by contact with the addictive substance. In psychological addiction, relief of pain is achieved by such as avoidance in phobias, obsessional thinking or compulsive acts. The addict becomes self-centered with a major goal in life that of relief of discomfort. This disorders interpersonal relationships and reduces achievements.

The Role of Endorphins

Addiction disorders methionine metabolism which evokes methionine enkephalin which is a self-made narcotic which maintains addiction. Also, in the same addictive process, leucine metabolism becomes disordered and produces leucine enkephalin, another self-made narcotic. The acid state that emerges during the withdrawal phase of addiction evokes opiate alkaloids which are another type of self-made narcotics. Addiction always involves a narcotic, but in the

case of addiction to foods and chemicals are self-made narcotics. Partially digested proteins can enter through the mucous membrane of the small intestine, arrive at the brain and evoke narcotic reactions. These are called exorphins and come from such as partially digested proteins such as gluten from the cereal grains or from beef or milk. Addiction is observed to suppress pancreatic function which in turn allows for the production of exorphins.

Disordered Amino Acid Metabolism

Likely, one of the most frequent disorders of amino acid metabolism is the loss of some inability to turn ammonia into urea. This provides a toxic state for the brain and can produce many mental symptoms and also muscular weakness. An amino acid profile reveals this to be a frequent occurrence. A less than optimum glutamic acid also provides a situation in which the brain cannot properly detoxify ammonia. This occurs because of a disordered citric acid cycle metabolism. It seems evident that these disorders by and large stem from addiction to food and chemicals but also in individual cases do have a relationship to genetic disorders of enzymes that handle amino acid functions. Secondarily through utilization demand, cofactors for the enzymes such as vitamins and minerals are also deficient. A primary nutritional deficiency would have a similar effect in disordering amino acid metabolism.

The Role of Approach and Avoidance

There are specific sets of neurones providing for the capacity of approach and the capacity of avoidance. The capacity to perform these responses are governed by specific synaptic junction transmitters which function adaptively at only optimum concentrations. Approach provides for the sociabilty to trust, to love and to achieve, while avoidance provides for protection against injury, mistrust and hostility. Either the function of approach or function of avoidance are operative at any single moment in time. Toxic states always evoke avoidance. Excessive synaptic junction transmitters can block approach response and activate avoidance response. Thus, it can be understood that a person reacting to a food or chemical or being otherwise in a toxic state can behave opposite to the way they have been taught. Thus, the person can when reacting to a food, hate the person they love.

The Sad Role of Tranquilizers

Tranquilizers have been observed to evoke methionine enkephalin, and in fact tranquilization is indeed narcotization. Therefore, tranquilizers are observed to be addicting due to this evoking of internally made narcotics. Based on this addictive quality of tranquilizers, they increase the incidence of maturity-onset diabetes. Tranquilizers should be reserved for refractory and treatment resistant cases and for those in whom the physiological determinants of their disordered behavior cannot be determined.

The Sad Role of Anti-Convulsants

Anti-convulsive medication is an absolute necessity for some patients, however they are known to make exceptional demands for nutrients and to sometimes interfere with development, especially of children. A large number of patients with seizures and nonseizure motor disorders can have their problem substantially solved by a bio-ecologic examination and treatment. Anti-convulsive medication should be reserved for the refractory cases.

SUMMARY

From ancient times, it has been observed that reactions do occur to environmental substances. Modern medicine has demonstrated by double-blind studies that reactions do occur to environmental substances. By and large, these are observed to not be allergies but rather be metabolic disorders stemming largely from a state of addiction but sometimes having genetic predispositions to these disorders.

Since prior existing metabolic disorders are demonstrated to predispose to maladaptive reactions to environmental substances, such cases logically should have the benefit of bio-ecologic examination and treatment, even though there will always be limitations of recovery. Present day laboratory assessment can do much to frequently pinpoint a specific vitamin, mineral or amino acid, the supplement of which will improve the condition. Bio-ecologic treatment needs to be integrated into other necessary therapies such as specific educational or psychotherapeutic needs. Bio-ecologic treatment is sufficiently rewarding that it should be given first place in the treatment of learning disorders, hyperkinesis, behavioral disorders, psychosis or seizures. Tranquilizers and anti-seizure medication should be reserved for refractory cases rather than used as the primary choice in such cases.

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APPENDIX

Clinical Ecology Double-Blind, Single-Blind, Animal Studies and Inappropriate Studies

Case I

Case II

Case III

Case IV

Case V

Toxic Methylated Amines

CLINICAL ECOLOGY
DOUBLE-BLIND, SINGLE-BLIND, ANIMAL STUDIES AND
INAPPROPRIATE STUDIES

Double-Blind Studies

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INAPPROPRIATE STUDIES WITH INAPPROPRIATE CONCLUSIONS

1. Judgment as to maladaptive reactivity based on one symptom only rather than multiple symptoms. Relatively fixed and singluar factor IgE immunology model mistakenly used for shifting multifactor and multisymptom nonimmunologic, metabolic disorders.

   Reference:
   Layman, C. W., Double-blind study of sublingual provocative food testing. A study of its efficiency. Ann Allergy, September, 1981; pages 144-149.

2. Studies inappropriately using the immunologic IgE model rather than the appropriate nonimmunologic metabolic model. The naturally occurring condition of metabolic homeostatic fluctuation of nonimmunologic reactions is ignored and or not understood, since the relatively fixed immunologic IgE model is being used.

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Note:
Experiments that do not have four to six days of avoidance before ingestion provocative testing are invalid due to the masking effect of food addiction. Experiments that retest ingestion provocative testing after 30 days of avoidance are invalid due to metabolic recovery potential of nonimmunologic reactions. Clinicians who judge immunologic (especially IgE) food reactions to be rare are correct. However, nonimmunologic metabolic maladaptive reactions to foods, chemicals and inhalants are frequent as judged by double-blind and single-blind provocative testing as well as by an assortment of metabolic abnormalities emerging in response to these evoked maladaptive reactions. Furthermore, these evoked metabolic abnormalities provide the most convincing evidence of the organicity of maladaptive reactions as well

as demonstrating the nonimmunologic cause of these metabolic disorders and the specific disease process involved in these reactions. For further elaboration of evidence, see:

The Roles of Bio-Ecologic Diagnosis and Treatment of Organic Factors in Mental Disorders. The Institute for Bio-Ecologic Medicine, P.O. Box 54589, Oklahoma City, Oklahoma 73154.

Bio-Ecologic Assessment of Reversible Metabolic Abnormalities in Epilepsy and Nonseizure Motor Disorders. The Institute for Bio-Ecologic Medicine, P.O. Box 54589, Oklahoma City, Oklahoma 74154.

CASE I

ECOLOGY HOUSE CLINIC AND LABORATORY, INC.
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Patient: Charlynne (Charlie)

INITIAL EXAMINATION AND HISTORY

Charlynne is a six-year-old white girl examined on 6-11-79. Her mother gave part of the history. Since age two she has had both Petitmal and Grandmal seizures. This is on a daily basis. Seizures especially occur when wakening. Petitmal seizures consist simply of a noding of the head, randmal seizures are violent, muscle contractures. Through the years she has been tried on dilantin, mysoline , phenobarbital, sodium valporate. No medication has controlled her seizures or seem to make any appreciable difference in the frequency of her seizure. She is observed to be hyperactive, have a short attention span and psychological testing is considered her to be borderline mentally defective. My observations were that she had a normal affect, that she was oriented in all spheres. The goal of examination was to determine the possible roll of ecologic factors and nutritional factors in her illness.

METHODS OF LABORATORY EXAMINATION

She was given meals of infrequent used foods for five days following which she was given meals of single foods with symptoms being monitored for after these meals as well as blood sugar one-hour after each meal. Cytotoxic testing was done. RAST testing. Intradermal testing. Hair test biopsy. After a stress day of eating foods that she was known to be reactive the urine was tested for the spillage of kynurenic acid and xanthurenic acid, formiminoglutamic acid. The next morning the serum was tested for insulin, vitamin, minerals, and amino acids. An immunologic survey, organ system functions was taken.

RESULTS OF LABORATORY EXAMINATION

Ten foods gave a blood sugar of 160 milligrams percent and beyond at one-hour. The highest reading was for corn which 310 milligrams percent. A large number of foods and chemicals gave her physical as well as central nervous system reactions. It appeared that there were seizures after some of the foods, however, the seizures were so frequently present that it was hard to be sure of the cause and effect of relationships. Hydrocarbons produced a trance like state. There were several foods that produced abnormal cytotoxic reations. Deficiencies were demonstrated in the hair and selenium in the blood and in zinc and chromium. In the blood amino acids were low. Especially noted was cystine and the essential amino acid phenylalanine as being low. There was a high spillage of kynurenic acid and xanthurenic acid in the urine indicative of a utilization problem of B-6. Formiminoglutamic acid was high revealing folic acid deficiency and the serum study for folic acid also demonstrated deficiency. Serum study demonstrated deficiency in Vitamin E. The urine study demonstrated deficiency in Vitamin C when reacting to grapes. Serum study

of magnesium revealed a deficiency. Alkaline phosphatase was high. Hyperthyroidism of an unspecified type was present.

FORMULATION

A cause and effect relationship between central nervous system malfunction and environmental substances both foods and chemicals was established. It was not established that the only cause for seizures were such exposures, however, it appeared evident that maladaptive reations do aid in initiating seizures. The deficiencies as noted in cystine which would cause a utilization problem in B-6, could predispose to seizures. The low folic acid would also, in a mild way, aid in the production of seizures. The folic acid deficiency may be somewhat a reflection of the utilization of anti-convulsive medication. Vitamin C deficiency would leave her in a state of toxicity which could, in turn, help produce seizures.

TREATMENT

She was placed on a four-day diversified rotation diet leaving out foods that produced either symptoms and/or high blood sugar. Appropriate nutrients of vitamins, minerals, and amino acids was supplied.

PROGNOSIS

This is guarded in view of the long term illness and in view of the inability to completely, specifically isolate an accurate cause and effect relationship to enviromental and seizures at all times. She should continue under close medical supervision, to assure a progressive attempt at solving the problem.

Patient: Charlynne (Charlie)

PRIMARY DIAGNOSES

1. 250.7 Chemica Diabetes Mellitus - Stress Type

A. Results of deliberate food testing of single food meals:

Food	Mg % blood sugar 1 hr. after meal
Dates	210
Figs	180
Raisins	160
Bananas	220
Cherries	180
Grapes	240
Cantaloupe	220
Corn	310
Beet. Sugar	160
Cane Sugar	160

2. 792.5 Allergy, Unspecified

A.

Food	Reaction
Dates	Very high energy, emotional, crying, very hyper, nervous, had light seizure at beginning of meal, 2 light seizures 15 min. after
Figs	LIght seizure 1 hour after meal
Raisins	Pulse change from 82 to 106 in 1 hr., hyper, crying, became very emotional 2 hours after eating, had 2 light seizures after starting meal, 1 light seizure ½ hr. after meal
Bananas	One seizure after beginning meal, a hard seizure 40 minutes later
Pineapple Juice	Pulse change from 80 to 105 in 75 min.
Fresh Plums	Pulse change from 98 to 112 in 60 min.. 1 seizure after meal
Cherries	Sleeping, 1 light seizure at beginning of meal, 1 light seizure an hour later
Oranges	Hard seizure at beginning of meal, 2 hard seizures an hour later
Canned Peas	Pulse change from 90 to 120 in 60 min., 2 light seizures while eating, another 20 minutes later, sitting sucking finger after meal

2. Allergy, Unspecified

Food	Reaction
Grapes	Hard seizure an hour after meal, before going to sleep, pulse change from 106 to 82 in 60 min.
Blueberries	Seizure at mealtime, 2 light seizures an hour later, hard seizure 50 minutes later
Prunes	Sleepy, hyperactivity, 2 light seizures while eating
Cantaloupe	More active, mild seizure, sucking finger
Rudabagas	Mild seizure, crying
Beets	Tense, very hyper, seizure
Tomatoe	Dark circles under eyes at night, puffy eyes, very hard seizure an hour after eating - loss of strength in limbs, mild seizure in 2 hours, hard seizure in 3 hrs.
Asparagus	Hopping around, hyper, seizure while eating, seizure 20 min. after
Acorn Squash	Seizure at beginning of meal?
Spinach	Seizure before & during meal?
Turkey	Mild seizure 2 hours after meal
Pork Chops	Hard seizure 2 hours later
Catfish	Light seizure 2 hours after meal
Tuna	Very high energy, very hyper - would not sit still, obstinate, disobedient - hitting & running around, seizure ½ hr. after meal, light seizure 15 min. later
Cod	Hard seizures after eating, 2 light seizures an hour later
Salmon	Two seizures after eating, became clumsy - falling around, disoriented, nervous, drowsy
Turbot	Two light seizures during meal
Monterrey Cheese	Two seizures
Cheddar Cheese	Hard seizure during meal
American Cheese	Two seizures after meal

2. Allergy, Unspecified

Food	Reaction
Milk	Crying, emotional, obstinate, hyper, pulse change from 92 to 141 in 90 min.
Millet	Acting tired and sleepy, irritable, crying
Wheat	Pulse change from 140 to 108 in 45 min.
Oatmeal	Seizure during meal, sitting upright with blanket, sucking thumb
Corn	Sleepy, seizure
Soybeans	Two seizures
Green Beans	Sleepy
Blackeye Peas	Sleepy, two mild seizure
Garbanzos	Two seizures after meal
Navy Beans	Tense, two seizures, sitting with blanket & sucking fingers
Peanut Butter	Calmer, less active, sleepy
Filberts	Pulse change from 116 to 80 in 30 min.
Almonds	One hard, one mild seizure
Walnuts	Two light seizures, two hard seizures
Egg	Hard seizure
Buckwheat	Three seizures
Honey	Sleeping, mild seizure
Cane Sugar	LIght seizure
Coconut	Jumping around, hyper, climbing on chair, pulse change from 95 to 130 in 30 min.

B. Inhalants - Hydrocarbon Petrochemicals

Reaction - Almost trance-like while sniffing, kind of sleepy, very still, eyes looked heavy

C. Cytotoxic - Rated 0 to 4

Substance	Reaction	Substance	Reaction
Chicken	trace	Grape Mix	trace
Coconut	trace	Cashews	trace
Olive Mix	trace	Apricot	trace
Cherry	1	Watermelon	trace
Peas	1	String Beans	1

C. Cytotoxic (cont)

Substance	Reaction	Substance	Reaction
Navy Beans	trace	Onion	trace
Wheat	trace	Bakers Yeast	1
Clover Honey	1		

SECONDARY DIAGNOSES

1. 305 Chronic nonpsychiatric Organic Brain Syndrome associated with physical conditions.
   Secondary to:

   A. Chemical Diabetes Mellitus
   B. Central Nervous System Allergic Reaction

Food	Reaction
Dates	Hyper, nervous, light seizure at beginning of meal, 2 light seizures 15 min. after
Figs	Light seizure
Raisins	Hyper, 2 light seizures after starting meal, 1 light seizure 30 min. after
Bananas	One seizure after beginning of meal, hard seizure 40 minutes later
Fresh Plums	Seizure after meal
Cherries	Slept, 2 seizures after meal
Oranges	Hard seizure at beginning of meal, 2 hard seizures an hour later
Peas (canned)	2 light seizures while eating, another 20 min. later
Grapes	Hard seizure after meal
Blueberries	Seizure at mealtime, then 2 light and one hard seizure
Prunes	Sleepy, hyperactive, 2 light seizures while eating
Cantaloupe	Mild seizure, more active
Rudabagas	Mild seizure
Beets	Tense, very hyper, seizure
Tomato	Very hard seizure an hour after eating with consequent loss of strength inlimbs, mild seizure and then a hard seizure

B. Central Nervous System Allergic Reaction

Food	Reaction
Asparagus	Hyper, seizure while eating and 20 min. later
Acorn Squash	Seizure at beginning of meal?
Spinach	Seizure before & during meal?
Turkey	Mild seizure 2 hours after meal
Pork Chops	Hard seizure 2 hours later
Catfish	Light seizure 2 hours later
Tuna	Very hyper, seizure after meal and 15min. later
Cod	Hard seizures after eating and 2 light seizures in two hours
Salmon	Two seizures after eating, became clumsy disoriented, nervous, drowsy
Turbot	Two light seizures during meal
Monterrey Cheese	Two seizures
Cheddar Cheese	Hard seizure during meal
American Cheese	Two seizures after meal
Milk	Hyper
Millet	Tired, sleepy
Oatmeal	Seizure during meal
Corn	Sleepy, seizure
Soybean	Two seizures
Green Beans	Sleepy
Blackeye Peas	Sleepy, two mild seizures
Garbanzos	Two seizures after meal
Navy Beans	Tense, two seizures
Peanut Butter	Sleepy, less active
Almonds	One hard, one mild seizure
Walnuts	Two light seizures, two hard seizures

B. Central Nervous System Allergic Reaction

Food	REaction
Egg	Two hard seizures
Buckwheat	Three seizures
Honey	Sleeping, mild seizure
Cane Sugar	Light seizure
Coconut	Hyper

C. Deficiencies

(1) 269.3 Minerals - results of hair biopsy

Selenium - 0 Normal Range - .04 - .12

Minerals - serum study

Zinc	10	Normal Range - 50 - 100 mcg/100ml
Chromium	1.0	Normal Range - 2.4 mcg/100ml

(2) 269.3 Amino Acid Assay - serum

13 absent
5 present in small amounts
Cystine is low
Phenylalanine is low

(3) 263.1 B6 - 24 hr. urine

Kynurenic Acid - 62MG Normal Range - up to 3
Xanthurenic Acid - 8.8 MG Normal Range - up to 3

(4) 263.8 Folic Acid
Formiminoglutamic Acid in urine -
Result: 7.9 MG 24/hr
Reference Range: under 3

Blood/Plasma Result: 2.3 ng/ml Normal RAnge: 5 - 24

(5) 266.8 Vitamin E

Blood/plasma Result: 0.6 Normal Range : 0.6 to 1.2

(6) 264 Vitamin C
Amount in urine 3 hours after meal of grapes, was - 5 mg/dl Normal Range: 40

2. Y72.9 Other Abnormal Blood Serum Findings

Substance	Result	Normal Range
Magnesium, Serum	1.44 MEQ/L	1.60 - 2.20

2. Other Abnormal Blood Serum Findings (cont)

Substance	Result		Normal Range
Total Bilirubin	0.07	MG/DL	.10 - 1.70
Alk. Phosphate	94	I.U./L	10. - 60
MCV	107	U3	80 - 103
MCHC	29.4	PCT	30 - 38

3. 244.9 Hypothyroidism - Unspecified

T3 uptake (% retention)	- 60	Normal Range:	43 - 60
T3 uptake (ratio)	- 1.2	" "	83
mcg. T-4/100 ml.	- 3.5	" "	4.7 - 10.15

CASE II

PHILPOTT FOUNDATION
PHILPOTT MEDICAL CENTER
William H. Philpott, M.D., Director
820 N.E. 63rd Street
Oklahoma City, OK 73105
405/840-4357

PATIENT: Kerri

INITIAL EXAMINATION AND HISTORY

Kerri is a seven year old white girl initially examined on 10-9-79. Her mother was present during the interview and gave the history. In April, 1979, she was reportedly diagnosed as having Tourette's Syndrome. The diagnosis of Tourette's Syndrome was based on her symptoms of grimacing, rhythmic mouth movements associated with rhythmic flexion of the arms and clenching of the fists, and generalized rigidity. Reportedly, she has episodic poor coordination and reversal of sequences. She is left-handed and has hyperextension capacity of joints. She has been attending a specialized class, and is not ready for the traditional first grade. As an infant, she had projectile vomiting. There was radiological evidence of an atonia of the pyloric valve. As an infant she was extremely underweight, listless, and had frequent ear infections with rupture of the eardrums. When milk, wheat and eggs were removed from the diet, ear infections subsided and she became alert. At age two, she was placed back on a full diet under the assumption of outgrowing these reactions to foods. By age three and a half she was manifesting odd grimacing of the face, associated with rhythmic clutching of the fists and flexion of the arms, and mouth movements. On neurological examinations she was suspected of having epilepsy. However, EEG was normal. Ann Warren, Ph.D., clinical psychologist, diagnosed her as hyperactive and attributed the grimacing to an attention-getting device. At age five she was examined by Lawrence Dickey, M.D., clinical ecologist, using sublingual testing technique, for examining reactions to foods, chemicals, and inhalants. Cane sugar as well as phenol evoked intense, bizarre, and rapid rhythmic facial grimacing, associated with flexion of the arms and clenching of the fists. Milk evoked hyperactivity. Corn evoked depression. Beet sugar evoked depression. Withdrawal from these foods evoked vomiting. Upon removal of these foods after three days she was happy, cheerful, and well-behaved with lessening of hyperactivity and grimacing. Soon her hair began to grow, which had been sparse since birth. She began to physically grow and her appetite improved. Grimacing and hyperactivity would still emerge under ill-defined circumstances. A four-day diversified rotation diet, initially leaving out the known reactors, was recommended, but not implemented. The known reactors were left out of the diet without a rotation diet being followed. At age six she was placed in a special developmental skills class. Her progress was inconsistent. At age six she was examined at Carrie Tingley Children's Hospital, Truth or Consequences, New Mexico, with the probable

diagnosis of cerebral palsy, and referred to Dr. Selinger, neurologist. Dr. Selinger gave her a diagnosis of Tourette's Syndrome, based on the rhythmic, facial, mouth, arm and hand movements.

My examination revealed her to be oriented in all spheres, with no psychotic ideation or behavior. A somewhat flat affect was observed. Her gait was normal during this initial examination, but was described by the mother as being episodically uncoordinated. Printing revealed reversal of sequences, wavy lines, and inconsistent size in the letters. Grimacing, arm flexion, and fist clenching was not present during the initial interview, but was later observed to emerge as rhythmic grimacing, arm flexing, fist clenching with mouth movements in response to specific foods. These movements were preceded by flat affect, social withdrawal, and lack of response to environmental stimuli.

METHOD OF LABORATORY EXAMINATION

She was given foods that she seldom used for a period of five days. Following this she was given meals of single foods, and monitored for the emergence of symptoms; especially writing was used to determine a loss of ability to write, poor coordination, reversal and so forth. In one 24 hour period she was given foods that she commonly used and the urine was collected for 24 hours and tested for spillage of kynurenic acid, xanthurenic acid, formiminoglutamic acid and vitamin C. The next morning the serum was tested for 12 vitamins, for minerals, and liver function tests, thyroid function tests and a battery of immunologic tests were done. Cytotoxic testing was done. A hair test biopsy was done.

RESULTS OF LABORATORY EXAMINATION

Ten foods were found to give blood sugars beyond 160 mg%. The highest was pineapple with a blood sugar of 230 mg%. Several had blood sugars of 200 mg% and beyond. Hypoinsulinism was demonstrated one hour after a test meal to a reactive food. A large number of central nervous system and physical symptoms were evoked due to reactions to the foods. RAST testing was positive to grass, to blue grass and ragweed. Intradermal testing was positive to sage brush, grass mix, household dust, dog, milk, cheese and Juniper. Chromium and zinc were deficient in the blood. Selenium was deficient in the hair. B6 utilization was demonstrated deficient in terms of high spillage of kynurenic acid and xanthurenic acid, whereas there was a normal amount of pyridoxine and pyridoxal-5-phosphate. Serum cystine was deficient. Vitamin C was deficient both in the blood as well as in the urine. Serum studies revealed a low IgG, and high IgM. A low C-4 fraction and a low Alpha-A-Acid Glycopro, low uric acid and a high alkaline phosphatase were also demonstrated.

FORMULATION

A cause and effect relationship was established between her foods and her symptoms. With a number of foods she would reverse the spelling of her name, reverse sequences, her writing deteriorated to a scrawl. Specific foods evoked a wide assortment of central nervous system symptoms including grimacing, tension, irritability, shaking, hyperactivity, lethargy, headache, silly affect, tiredness, muttering to self, aggressive acting out, tongue rolling, confused thinking and the emergence of depression and crying. The deficiencies of vitamins would help to produce these symptoms, but they may well be in response to the excessive demand the maladaptive reactions to foods and chemicals make.. B_6 was normal as judged by serum pyridoxine and pyridoxal-5-phosphate but utilization of B_6 was abnormal as judged by the excessive spillage of kynurenic acid and xanthurenic acid. Such a disordered utilization of B_6 is characteristic of and can therefore be explained by the deficiency of cystine. Cystine deficiency would be consistent with a metabolic disorder of conversion of methionine to cystine. An early diabetes mellitus disease process was evidenced by 10 foods with hyperglycemia at one hour post meal of single foods. These hyperglycemic reactions ranged up to 230 mg. % at one hour post test meal. Hypoinsulinism was present again confirming the stage of chemical diabetes.

TREATMENT

She was placed on a 4 day diversified rotation diet, leaving out the foods that evoked hyperglycemia and/or symptoms. These could be returned to the diet in 3 months if they do not produce symptoms or hyperglycemia. She was placed on appropriate vitamins, minerals, amino acid supplements. She should be seen at 6 month intervals to insure a successful program.

PROGNOSIS

The prognosis is good. The motivation is high for following the program. She will be dependent on the adults in her environment to provide the correct motivation.

William H. Philpott, M. D.

PATIENT: , Kerri

PRIMARY DIAGNOSES:

1. 250.7 Chemical Diabetes Mellitus - Stress Type
   A. Results of deliberate food testing of single food meals:

Food	Mg% Blood Sugar 1 hr. after meal
Raisins	225 mg%
Peach	170 mg%
Grapes	190 mg%
Rice	200 mg%
Honey	170 mg%
Pineapple	230 mg%
Bananas	195 mg%
Sweet potatoes	165 mg%
Dates	200 mg%
Papaya	200 mg%

   B. Hypoinsulinism
   1 hr. after test meal of beef - blood sugar 55 mg%
   Micro insulin 34 U.ml normal 60 or more U.ml

   1 hr. after test meal of raisins - blood sugar 225 mg%
   Micro insulin 60 U.ml normal 60 or more U.ml

   This is borderline hypoinsulinism in which insulin was not adequate to prevent rise of blood sugar to hyperglycemic level.

2. 292.5 Allergy, unspecified
   Results of deliberate food testing of single food meals:

Food	Reaction
Plums	Burping, rubbing nose while talking, constantly moving her hand, keep repeating words
Orange	Pulse drop of 40 beats at 75 min.
Pineapple	Pulse rise of 40 beats in 30 min. (80-120), very talkative and very hyperactive, after 60 min. became extremely tired, slept
Peach	Eyes itching, fussy and frustrated, crying, depressed, later began to grimace, handwriting reflected depression with letters smaller, more closely spaced and inconsistent in size, pulse rise of 16 (76-92)
Bananas	Pulse rise of 24 in 1 hr., circles under eyes, grimacing
Grapefruit	Arm aching
Honeydew melon	Eyes itching, grimacing
Cantaloupe	Stomach ache and diarrhea

PATIENT: Kerri

Blueberries	Hiccoughs
Grapes	Nose and eyes itching, feels like vomiting, tired, layed on couch, grimaced, pulse rise of 20. (80-100)
Corn (mature)	Difficulty reading, tongue rolling, grimacing.
Dried papaya	Hyperactive, tongue rolling, complete reversal of letters when writing name, writing was scrawly. Pulse rise 48 pt. (72-120)
Figs	Grimacing and hyperactive.
Pears	Eyes itching.
Broccoli	Stomach cramps soon after eating.
Carrots	Pale, grimacing while drawing pictures.
Sweet Potato	Violent headache and vomited up potato.
Green pepper	Feels like vomiting, gas in stomach.
Celery	Diarrhea, Nose itching.
Potato	Tongue rolling and biting, grimacing, hyperactive, eyes itching, handwriting scrawly, picture drawing deteriorated to scrawl. Inability to remember and sequence ABC's. Pulse rise of 18. (84-102)
Salmon	Diarrhea.
Cashews	Sick, nauseated.
Pecans	Pulse rise of 24 in 75 min. Hiccough, eyes itching, feeling tired.
Cheddar Cheese	Palor, pulse increase of 24, slightly hyperactive.
Cottage Cheese	Rubbing nose continually, grimacing while playing, hyperactive, impatient, demanding, complaining of leg being tired, frustrated because of trouble drawing a picture handwriting was shakey at 60 min., unable to remember sequence of ABC's. Pulse rise of 20 pt. (96-116)
Yogurt	Tongue rolling, gray, ashey color, talking to self, dark circles under eyes.
Cheddar Cheese	Slightly hyperactive, grimacing. Pulse rise of 24. (100-124)

PATIENT: Kerri

Milk	Rubbing nose, hyperactive, frequent urination. Pulse rise of 20 pt. (100-124)
Pork	Hyperactive, handwriting scrawly.
Beef	Pale, energy low, expressed trying not to grimace.
Peanuts	Grimacing, palor, pulse rise of 20 pt. (88-108)

Inhalants

Somewhat paler after sniff test of petrochemicals.

RAST

Results positive on: blue grass and ragweed.

Intradermal

Results positive on: Sagebrush, grass mix, house dust, dog, milk, cheese and Juniper.

PATIENT: Kerri)

SECONDARY DIAGNOSES

1. 305 Chronic Nonpsychotic Organic Brain Syndrome

Secondary to:

A. Chemical Diabetes Mellitus

B. Central Nervous System Allergic Reaction

Food	Reactions
Plums	Constantly moving her hands, repeating words.
Pineapple	Very talkative, more hyperactive.
Peach	Depressed, fussy and frustrated, crying.
Honeydew	Grimacing.
Grapes	Lethargic.
Apple	Acting silly.
Figs	Hyperactive.
Sweet Potato	Violent headache.
Potato	Hyperactive.
Pecans	Tired.
Cottage Cheese	Hyperactive, impatient, demanding.
Cheddar Cheese	Hyperactive.
Milk	Hyperactive.
Yogurt	Grimacing, talkative, muttering to herself.
Pork	Hyperactive.
Beef	Low Energy.
Millet	Hyperactive, whiny, sullen, kicking table.
Lentils	No energy.
Black olives	Feels hot, oral temp. up 1°.

PATIENT: Kerri

Raisins	Grimacing, whiny.
Peanuts	Grimacing.
Dates	Perception problems, impatient.
Peas	Silly, depressed, impatient, grimacing, tongue rolling.
Chickpeas	Tired, frustrated.
Cane Sugar	Crying and fussy, impatient, bad disposition, pulse rise of 28 pts.
Dates	Impatient, confused thinking.
Sunflower Seeds	Whiny, impatient, grimacing, bedwetting.
Mushrooms	Pouty, impatient.
Eggs	Sleepy, feels mean and destructive.
Honey	Irritable, hitting table.
Lactose	Shakey, writing very poor.
Wheat	Grimacing.

C. Deficiencies

(1) 269.3 Minerals - Results of hair biopsy

	Results	Normal Range
Low Selenium	0	.04 - .12
High Calcium	115	46 - 85
High Magnesium	14	6 - 10.1
High Zinc	21	13 - 20
High Phosphorous	20	9 - 15

(2) 269.3 Minerals - Serum

	Results	Normal Range
Zinc	48	50 - 100 MCG/100 MLS
Chromium	1.0	2.4 MCG/100 MLS

PATIENT: Kerri

(3) 263.1 B6 - 24 hr. Urine

	Results	Reference Range
Kynurenic Acid	26 MG/24 hr.	Up to 3
Xanthurenic Acid	9.8 MG/24 hr.	Up to 3

(4) 264 Vitamin C - Urine

0 amount Ascorbic Acid in urine 3 hours after meal of rice with blood sugar of 200 mg%

264 Vitamin C - Blood plasma

0.1 mg% Normal Range 0.4-1.5

(5) Amino Acids - Serum

Deficient essential amino acids

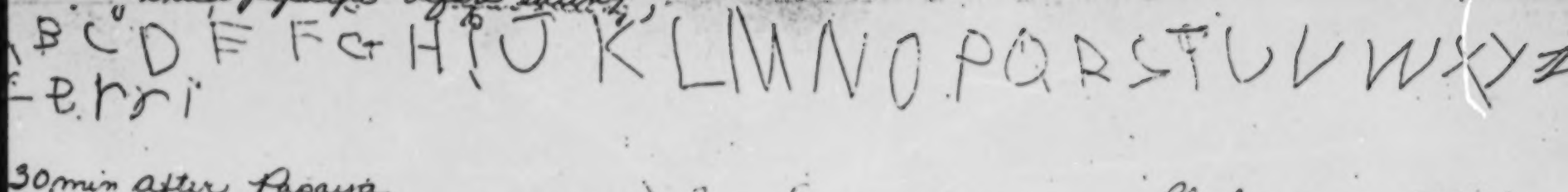
	Results	Reference Range
Isoleucine	3 MCM/100 ML	3.5 - 10
Leucine	6 MCM/100 ML	6.9 - 16
Lysine	8 MCM/100 ML	9.0 - 26

Deficient nonessential amino acids

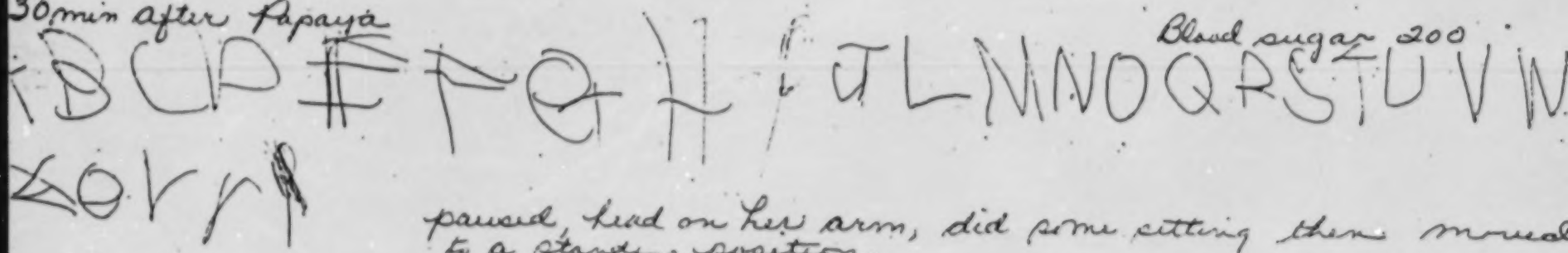
	Results	Reference Range
Cystine	present	3.1 - 14
Aspartic Acid	present	1.1 - 5.4
A-amino-N-Butyric Acid	present	0.8 - 3.5

Excess amino acids

	Results	Reference Range
Glutamic Acid	17 MCM/100 ML	0 - 12
Alanine	53 MCM/100 ML	17 - 15



30 min after Papaya

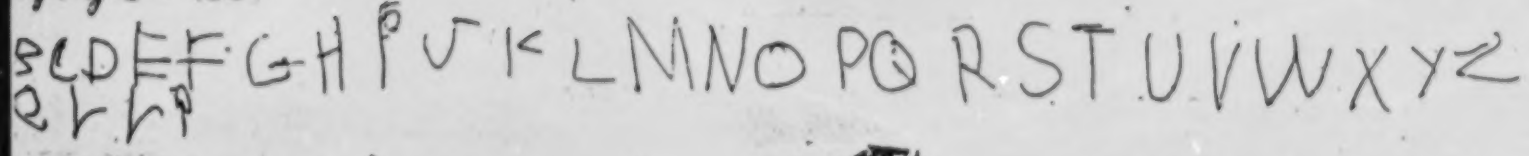


Blood sugar 200

paused, head on her arm, did some sitting then moved to a standing position

became hyped, more talkative lots of tongue rolling, yawning, pale, feeling sleepy.

Papaya 1 hr.



Thanks for your support and being so kind to Terri & I.

Love

Terri

38 This dyslectic writing was seen after eating large quantities of dried papaya

NOTE: At 30 minutes post papaya test meal, her writing deteriorated, K & P were left out of the alphabet, tongue rolling, hyperactive, sleepy and a blood sugar of 200 mg% (normal post-meal blood sugar is below 160 mg%). Soon after this, she mirror image signed her name.

Kerri Six Month Follow-up Report
December 1981

Age 8

The following tests were ordered:

1.	Hair Analysis	Results below
2.	CBC	Normal
3.	Cytotoxicity	Results below
4.	Candida species	Normal
5.	Chemistry screen	Results below
6.	Total iron	Normal
7.	Glycohemoglobin	Normal
8.	Vitamin Profile	All normal
9.	Xanthuranic & Kynurenic acid	Normal
10.	Amino acids (24 hrurine)	Results below

269.3 Minerals - Results of hair analysis shows:

	Results	Reference Range
Low calcium	33	46-85
Low Potassium	2	6-23
Low Iron	1	1.5-5
Low Manganese	.04	.05-.17
Low Chromium	.01	.04-.07
High Phosphorus	20	9-15

Note serum iron was normal range.

277.9 Unspecified disorders of metabolism as manifested in cytotoxic testing. Graded 0-4

Grade #1 - Floride
Grade TR - Milk, Roquefort cheese, strawberries, cashews, apricots, tuna, halibut, tomatoes, onions, cabbage mix, beets, carrots, wheat, corn, rice, coffee and cinnamon.

270.9 Amino Acid deficiencies and disorders.

Results of 24 hr urine collected 12-1-81.
Stressor foods plus - 2 Gm Histidine 1½ Gm Tryptophane
1 Gm Methionine

Essential Amino Acids	Results	Reference RAnge
Valine	9 MCM/24hr	15-40
Isoleucine	Tr MCM/24hr	20-200
Leucine	7 MCM/24hr	21-200
Methionine	55 MCM/24hr	20-50
Other Low Amino Acids		
Glutamic Acid	8 MCM/24hr	40-150

Kerri
December 1981

The Following are above Reference RAnge:

Methionine Sulfoxide	7 MCM/24hr	0-Tr
Gamma-AminoButyric Acid	71 MCM/24hr	0-Tr
Arginine	45 MCM/24hr	0-30

Other Abnormal Findings:		Reference Range
Creatinine	0.6 MG/DL	0.8-1.2
(Note: Alkaline Phos was 251 1U/L May be normal for child).		35-148

J.B. Pangborn, Ph.D.
President

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Medical Director

James Chambers, Ph.D.
Consulting Biochemist

Bionostics, Inc.
P.O. Drawer 400
Lisle, Illinois 60532

January 26, 1982

"Specializing in Biochemical Interpretations of Amino Acid Analyses"

Phone: 405/840-4968

Kerri

B-00-24-82

Laboratory Data: Amino acid analysis including quantitative 24 hour urine amino acid analysis, Bio-Science Laboratories; hair analysis Automated Hair Analysis Laboratory; vitamin analysis, New Jersey Medical School Vitamin Assay Center; other laboratory data from Philpott Medical Center, Oklahoma City, OK.

Referring Physician: William H. Philpott, M.D., Philpott Medical Center, Oklahoma City, OK

Introduction

The interpretations presented in this report are made from the viewpoint of amino acid chemistry and use as a primary basis the laboratory analysis(es) for urine amino acid content. Supporting information consists of: the blood vitamin analysis (to infer whether a coenzyme is present in sufficient quantities), the hair and blood mineral analyses (to infer whether a mineral activator for an enzyme is likely to be present in proper abundance), and the doctor's written report of examination which may provide descriptions of symptoms that are known to be associated with specific amino acid disorders. Other laboratory results may also be useful. The strategy behind the interpretation is to identify biochemical disorders or deficiencies that potentially are common

to two or more anomalies in the amino acid analysis. Several anomalies with the same potential cause tend to confirm the existence of the causative disorder. Diagnoses thus are suggested by the evidence but are limited in scope to the amino acid information on which they are primarily based.

Analytical Observations

Urine analysis shows three of the ten essential amino acids to be low.

Disorder in transamination type reactions is indicated by gamma-aminobutyric acid and possibly by methionine sulfoxide.

High alkaline phosphatase and slightly high lactic acid dehydrogenase may indicate tissue destruction.

High histidine can indicate histidinemia.

Observed are the following:

1) Incomplete protein digestion and/or poor protein diet and/or poor absorption of amino acids (digested proteins) is indicated by three of the ten essential amino acids being low. These three essential amino acids are valine (9), leucine (7), and isoleucine (trace). Hair analysis for manganese (0.04) is low.

2) Metabolites indicating disorder in transamination reactions are gamma-aminobutyric acid (71) which is high and methionine sulfoxide (7) which is high.

3) High alkaline phosphatase (251) and slightly high lactic acid dehydrogenase (135) might indicate tissue destruction.

K.

4) Creatinine (0.6) is low.

5) Histidine (1823) is high.

6) Glutamic acid (8) is low.

7) Minerals

	Hair	Blood
Calcium	(33) low	normal
Potassium	(2) low	normal
Phosphorous	(20) high	normal
Iron	(1) low	normal
Manganese	(0.04) low	
Chromium	(0.01) low	

8) Vitamins: The following vitamins are all within the normal range: vitamin B_{12}, folic acid, vitamin B_6, thiamin, nicotinamide, biotin, riboflavin, pantothenic acid, vitamin C, vitamin E, vitamin A, and beta-carotene.

Discussion

Incomplete Proteolysis

Incomplete proteolysis during protein digestion and/or poor protein diet and/or poor absorption of amino acids (digested proteins) is indicated by three of the ten essential amino acids being low. These three essential amino acids are valine, leucine, and isoleucine. All of these essential amino acids are used in the preparation of proteins. Some of these proteins are enzymes which serve as catalysts to enable the chemical reactions throughout the body to occur. Another use of proteins is as antibodies (immunoglobulin). The essential amino acids can also be used to prepare proteins. Some of the essential amino acid metabolites have specific functions such as producing serotonin, a

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neurotransmitter from tryptophan.

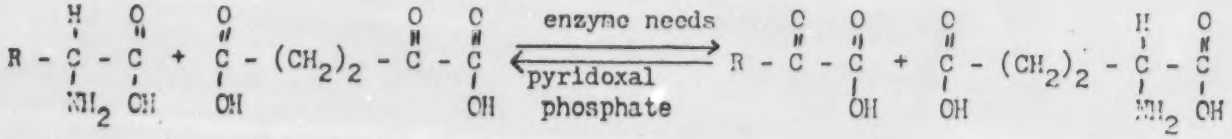
Complete digestion of proteins requires the secretion of proteolytic enzymes into the digestive tract. These enzymes, to be effective, must be in a medium of proper pH (not too acidic or too basic) and also manganese and zinc are needed to activate these enzymes. The hair analysis for manganese is low for this patient.

The three low essential amino acids are simular in that their carbon atom structure is an open chain (not cyclic) and branched. None of the other essential amino acids have this open chain branched structure. Could it be that this patient has difficulty in absorbing open chain branched structure amino acids through the intestinal walls?

Transamination

There are several indications of malfunction in reactions involving pyridoxal phosphate (from vitamin B_6) and/or glutamic or alpha-keto-glutaric acids. Glutamic acid deficiency is to be expected since it is low.

Transamination reactions change an $-\overset{H}{\underset{NH_2}{C}}-$ (amino) group in a molecule to a $-\overset{O}{\overset{\|}{C}}-$ (keto) group and the reverse of this occurs in another molecule. The other molecule is alpha-ketoglutaric acid that contains a keto group and which goes to glutamic acid that contains the amino group as shown in the equation.

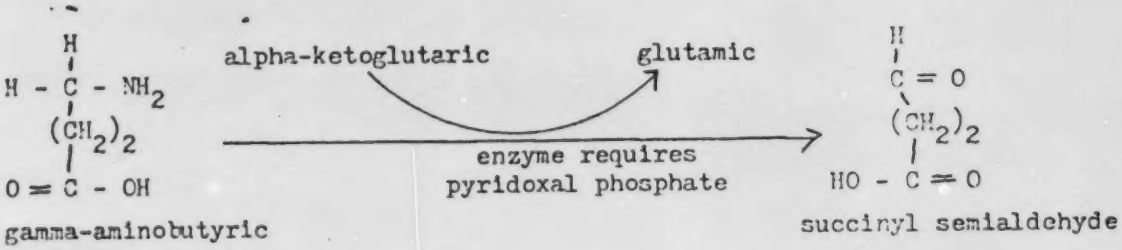


alpha-ketoglutaric

glutamic acid

Note the above is a reversible reaction and can go either way depending on the body's need. For this reaction to occur pyridoxal phosphate (from vitamin B_6) and alpha-ketoglutaric acid or glutamic acid are needed depending on the direction of the reaction. This patient has a low glutamic acid value.

Gamma-aminobutyric acid is high, which indicates a deficiency in the following transamination reaction.



gamma-aminobutyric

succinyl semialdehyde

A deficiency in alpha-ketoglutaric acid and/or pyridoxal phosphate would slow down the above reaction causing gamma-aminobutyric acid to increase. Glutamic acid is produced from alpha-ketoglutaric acid, therefore, a low alpha-ketoglutaric acid could be the cause of the low glutamic acid.

There is a possibility that the high methionine sulfoxide is due to a transamination malfunction. Note the following equations.

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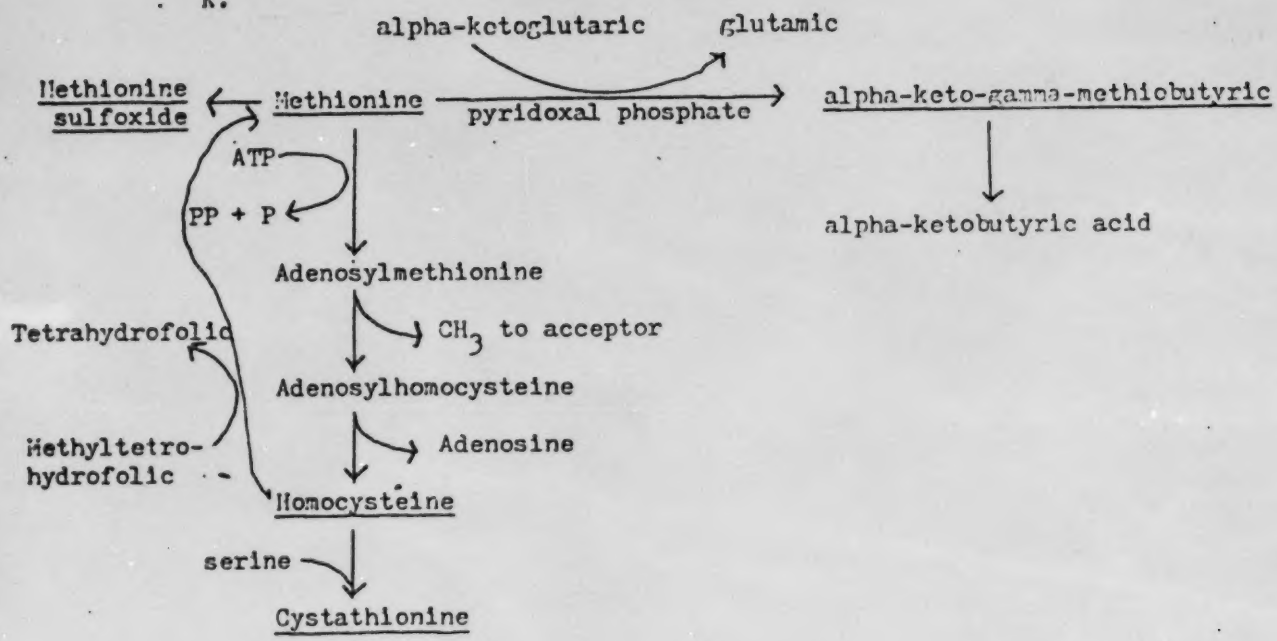

From the above equations it can be seen that methionine can undergo a series of reactions to homocysteine and cystathionine, also by a transamination reaction methionine can go to alpha-keto-gamma-methiobutyric acid and alpha-ketobutyric acid, as well as going to methionine sulfoxide. A malfunction in homocysteine going to cystathionine will cause more homocysteine to go back to methionine. Under these conditions (hindered metabolism of methionine) methionine has been known to produce excess methionine sulfoxide. With this patient this series of reactions seems to be functioning normally. If there is a malfunction in the transamination reaction (methionine to alpha-keto-gamma-methiobutyric acid) this could slow the reaction and it would appear this would also hinder the metabolism of methionine and cause more of it to go to methionine sulfoxide.

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Tissue Destruction

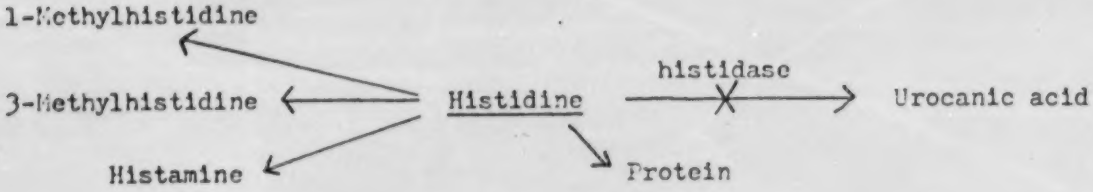
A high value for alkaline phosphatase and slightly high value for lactic acid dehydrogenase could indicate tissue destruction. These two substances are enzymes which are within tissue and if there is tissue destruction they can be released and their value would thus increase.

Creatinine

Creatinine is low. Creatinine is produced in muscle tissue. This patient is a seven year old girl and if she has small muscles this may be the reason for a low creatinine value.

Histidinemia

Histidine is high. An important reaction of histidine is histidine to urocanic acid. See the following equations.



There is an inborn disease in which the enzyme histidase, which enables histidine to go to urocanic acid, is either absent or is weak. This causes histidine to increase. This histidase disorder is known as Histidinemia. It appears that there is no single abnormality that is common to all histidinemia patients. Abnormalities that often occur with histidinemia are slow development both mentally and physically.

slurry speech, and frequent infections. Have there been times when this patient was under-weight and slow in developing both physically and mentally?

Probable Diagnoses and Suggested Supplements

Probable

1) Poor protein digestion and/or poor protein diet and/or poor absorption of amino acids is indicated by three essential amino acids with a low value. This may be due to a problem of poor absorption of valine, leucine, and isoleucine through the digestive tract.

2) A malfunction of some transamination reactions as indicated by the metabolites gamma-aminobutyric acid and methionine sulfoxide.

3) Slight chance of tissue destruction as indicated by the enzymes alkaline phosphatase and lactic acid dehydrogenase.

4) There is a slight possibility of histidinemia.

Suggested Supplements

Amino acids: valine, leucine, isoleucine, and glutamic acid. Keep histidine adequate but not in excess.

Enzymes: proteinases

Minerals: calcium, potassium, iron, manganese, chromium.

Vitamins: continue with any present program.

Disclaimer

This interpretive report is a written consultation to the physician

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and is not a recommendation for treatment. The stated diagnoses are considered probable or possible based on the submitted biochemical data, and the supplements are suggested on that basis also. It is the physician's prerogative to prescribe treatment considering this consultation and other medical data known to him.

Distribution:

3 cys: Dr. Pangborn (2 for Dr. Philpott)

orig: Dr. Chambers

James R Chambers

James R. Chambers, PhD

Reviewed Jan 31, 1982

J W Pangborn Ph.D.

CASE III

PHILPOTT FOUNDATION
PHILPOTT MEDICAL CENTER
William H. Philpott, M.D., Director
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Oklahoma City, Ok. 73105
405 / 840-4357

Michael

1-8-81

INITIAL EXAMINATION AND HISTORY

Michael is a seven year old boy examined on 1-8-81. His mother was present during the interview. His chief complaint is that he has a learning disability that has been diagnosed as having defective motor skills, dyslaxia, and hyperactivity. A cat scan is indicative of some degree of cerebral atrophy. His IQ is in the 70's. However, he does not concentrate well enough on the test to give his full potential. He tells of seeing two objects where there is only one. This happens especially in the morning and leaves later in the day. He also usually has pain behind the knees in the morning which tends to leave later in the day. At birth he had a difficult time because he had a staph infection of both the nose and the umbilicus and had to have antibiotics. He was milk allergic and would have generalized edema in response to milk. No formula was found that he could use. He was not able to tolerate soy as well as dairy products. He was given juices and cereals from the first. Lately there has been swelling of his face and eyes which is episodic. Hives have been noted on his face. He is very sensitive to heat and cold. He tends to keep a coat on. He is sensitive to bright lights and noise. He has night sweats. He grits his teeth at night. He sleeps excessively sound. As an infant he was prone to ear infections. He wore braces because his legs turned out due to a hip deformity. He had a candida albicans infection of the throat at five months. He has always been hyperactive and has a short attention span. He transposes letters. He attends a special school for learning disabilities. My observations are that he is very hyperactive, very curious and quite mechanically inclined and capable of figuring things out. His affect was quite normal. He recently underwent an allergy study and was found to be allergic according to skin tests to a high number of foods and chemicals, and enviromental substances. He has not received an injection therapy program. The mother has elected to have an ecology examination made and make optimum use of avoidance, especially the area of foods.

SHAWN
February 8, 1981

DSM III MULTIAXIAL EVALUATION

Axis I.
314.01 Attention Deficit Disorder
with hyperactivity (hyperkinetic)

312.10 Conduct Disorder

The following foods tested evoked hyperactivity, violent actions, crying, anger, aggressiveness, belligerance and rebellious of parental authority:
beets, rutabaga, pineapple, cantaloupe, peaches, carrots, apples, watermelon, oranges, turkey, chicken, fresh corn, oats, cashew nuts, peanuts, peas, lima beans, milk, American cheese, cheddar cheese, cottage cheese, Monterey Jack cheese, maple sugar, buckwheat and honey.

Axis II.
315.00 Reading Disorder, developmental
Short attention span - attends learning disability school

Axis III.
579.0 Celiac Disease
1-13-81 Results of alkaline phosphatase - 181 IU/L
1-14-81 Results of alkaline phosphatase - 164 IU/L
Reference range 35-148
This shows a drop of 17 points.

790.2 Chemical Diabetes Mellitus - stress type
Results of deliberate food testing of single food meals

A. Mg% blood sugar one hour after each meal:
rutabagas - 172 mg% asparagus - 200 mg%
figs - 194 mg% honeydew melon - 205 mg%

B. Ketones in urine - 4+

C. Small intestine pH was 6
normal 7.5-9
Low bicarbonate production, therefore presumed low enzyme production.
This testing is consistent with chemical diabetes.

269.2 Vitamin Deficiencies

B6 utilization disorder - results of 24 hour urine shows
kynurenic acid to be 8.8 MG/24hr
xanthurenic acid to be 5.9 MG/24hr
reference range up to 3

Vitamin C
ascorbic acid in urine was 5 mg/dl
normal 40

Folic acid by paramecium study
4.3 ng/ml
normal 5.0-24.0

269.3 Mineral Deficiency - results of hair analysis

	Results	Normals
low zinc	12 mg%	13 - 20
high magnesium	10 mg%	3 - 7
high copper	4.6 mg%	1.1 - 3.1
high chromium	.16 mg%	.04 - .07

Toxic metals - results of hair analysis

cadmium	.16 mg%	.03 - .11
lead	1.7 mg%	.4 - 1.1

270.9 Amino Acid Deficiencies and Disorders

Results of 24 hour urine collection made 1-13-81
Stressed with commonly used foods plus 10 grams tryptophan, 10 grams histidine and 3 grams methionine

Essential amino acids	Results	Reference range
valine	absent	14 - 51
leucine	trace	21 - 200
cystine	absent	20 - 130
threonine(borderline low)	85 MCM/24hr	85 - 440
phenylalanine(borderline low)	24 MCM/24hr	24 - 190

Below reference range	Results	Reference range
a-aminoadipic acid	trace	11 - 70
taurine	47 MCM/24hr	63 - 2300
1-methylhistidine	68 MCM/24hr	130 - 930

Above reference range	Results	Reference range
homocitrulline	trace	0
hydroxyproline	149 MCM/24hr	0

270.8 Specified Disorder - Hydroxyprolinemia

277.9 Unspecified Disorder of Metabolism
Errors of the metabolic process as manifested in reactions to:

A. Foods
beets - cheeks red, ear itch, yawning
tomato - stomachache
eggplant - legs hurt
carrots - legs hurt too bad to walk, tired
lettuce - flushed, headache
cauliflower - stomachache, listless, legs hurt
okra - acts drugged, red cheeks, disagreeable
turnip greens - tired, stomachache
squash - red cheeks, seeing double
brussel sprouts, apple, turkey, mature corn, tuna, shrimp, lobster, mushrooms, lactose and pumpkin seeds - stomachache
asparagus, lobster and coconut - legs hurting
beef - backache
lettuce, pecans and peas - headache
raisins, maple and duck - diarrhea
peaches, chicken, Brazil nuts, lobster and peanuts - double vision

B. Cytotoxic test - graded 0 to 4
grade 2+ - chicken
grade 1+ - roquefort cheese, navy beans
grade 1 - pork, lamb, lettuce, buckwheat, floride
grade trace - grape mix, sole, rice, barley, clover honey, chlorine

C. Reactions to Inhalants
hydrocarbons - sleepy, emotional, cried easily
ammonia - very tired, seeing double

796.4 Other Abnormal Lab Findings
Results of hematology

	Results	Normals
RBC	4.12	4.6 - 6.2
HCT	35.8	42 - 54
HGB	12.5	14 - 18

Darkfield Study - rating 0-4
result was 2

Axis IV. Psychosocial stressors - non related to condition

Axis V. Highest level of adaptive functioning past year - poor

SHAWN
February, 1981

FINAL FORMULATION

He has been allergic to foods since infancy. He was highly allergic to milk and soy, therefore he was started on cereal grains immediately. There is clinical evidence of generalized facial edema in response to reactions to environmental substances.

Provocative testing revealed a cause and effect relationship between his behavior and a wide assortment of foods. Celiac disease was demonstrated by a drop in alkaline phosphatase on an exposure to wheat. Several foods produced a high blood sugar, revealing evidence of a chemical stage of diabetes mellitus. Vitamin deficiencies were B6, vitamin C and folic acid. Mineral deficiency as determined by the hair test was low zinc. Heavy metals according to the hair test that were higher than normal were cadmium and lead. Amino acid study reveals incomplete lysis, or the other alternative is poor absorption of amino acids once they are digested. It may well be that both exist. For details of the amino acid study and recommendations, see Dr. Jon Pangborn's report of April 9, 1981.

FINAL RECOMMENDATION

He should follow carefully a four day diversified rotation diet initially leaving out foods that produce symptoms and or hyperglycemia. These may be returned in three months on a once in four day basis if they do not produce symptoms and or hyperglycemia. Specific supplements of vitamins, minerals and amino acids should be arranged according to the laboratory findings. There should be frequent follow-up interviews to assure success. It would be best if he were followed in the office on a six months review basis for laboratory reassessment.

Name: Shawn

Jon B. Pangborn, Ph.D.
April 9, 1981

Laboratory Data: Quantitative 24 hour urine amino acids, Bio-Science Labs, 1-14-81

Referring Physician: William Philpott, M.D. Philpott Medical Center
Oklahoma City, OK

Analytical Observations

The urine amino acid distribution of this patient showed curious abnormalities--most essential amino acids were low or deficient yet certain amino acids and their catabolism products showed moderate presence. A telephone conversation with Dr. Philpott on April 2nd revealed that a loading of 10 gm tryptophan, 10 gm histidine, and 3 gm methionine had been administered; tryptophan to stress B_6 utilization, histidine to stress H_4-folate utilization, and methionine to stress possible disorders of the transsulfuration pathway and B_6 utilization.

Observed are:

1) tryptophan catabolism products: higher than normal kynurenic (8.9) and xanthurenic (5.9) acids

2) histidine catabolism/metabolism products: normal formiminoglutamic acid (1.4, FIGLU), normal 3-methylhistidine (63), lower than normal 1-methylhistidine (68), low but acceptable glutamic acid (68)

3) methionine catabolism products: normal methionine (58), normal (absent) homocystine, normal cystathionine (22), and abnormal (absent) cystine, and low taurine (47).

The deficient essential amino acids are argenine (absent) isoleucine (38, borderline low), leucine (present), lysine (60, borderline low), phenylalanine (24), threonine (85), valine (absent). The acceptable-level essential amino acids were all "loaded" in.

Discussion

Since there is no evident blockage at the alpha-aminoadipic acid step above alpha-ketoadipic acid (where the lysine and tryptophan pathways converge), the presence of excess kynurenic and

xanthurenic acids seems singularly indicative of a pyridoxal phosphate deficiency. There is no evidence of metabolic blockages due to enzyme deficiencies in the lysine or tryptophan catabolism pathways. However, the lysine supply to its path was so low that the enzymes were hardly tested.

The histidine pathway appears to operate satisfactorily, and H_4-folate is not implicated as deficient.

The methionine pathway shows anomalous catabolism since methionine, homocystine and cystathionine are normal, yet cystine (and probably cysteine) is absent and taurine is low. When cystine does not show at all ("absent" versus "present"), one should worry about the insulin balance and the glutathione level. (Cysteine is a key amino acid in the A and B chains of insulin which are actually joined by the -S-S- bond between cysteine fragments in the insulin protein.)

The metabolic blockage or enzyme deficiency is not apparent from the data. The absence of homocystine implies absence of homocysteine, therefore cystathionine beta-synthase and pyridoxal phosphate are operative. The normal value of cystathionine (22) implies normal cysteine formation and ammonia release with alpha-ketobutyrate formation in the presence of cystathionine gamma-lyase, but cystine is completely absent. It is possible (but not likely) that the absence of the oxidized dimer forms, homocystine and cystine, do not reflect the absence of the corresponding reduced amino acids. Cytochrome oxidase is reported to be the enzyme for cystine formation from cysteine (Gerritsen and Waisman, "Homocystinuria", Chapter 21 of The Metabolic Basis of Inherited Disease, Stanbury, Wyngaarden and Fredrickson, 3rd ed., p.420.)

Another possibility is that cysteine and cystine are essentially completely converted to other metabolits using all available major and minor pathways. Driving this is the general deficiency of amino acid metabolites stemming from the lack of uptake in essential amino acids. Cystine can be transformed into: cysteine, cystine disulfoxide, thiocystine, pyruvic acid; cysteine can be transformed into cystathionine, cystine, glutathione, mercapto-pyruvate, pyruvic acid, systeamine, hypotaurine, taurine, cysteine sulfinic acid, and alanine plus sulfite. Once cysteine becomes pyruvic acid, it is two steps away from the citric acid cycle via acetyl-CoA.

The major problem with Shawn's amino acid catabolisms and metabolisms is that the necessary feedstock or nutritionally essential amino acids are not entering the organs (primarily the liver) where the catabolism/metabolism sequences would normally occur. Since the loaded essential amino acids and their catabolites are present, mostly in normal amounts, the root cause appears to be either (a) lack of protein breakdown during digestion or (b) lack of amino acid uptake and transport through the intestinal mucosa and into the blood.

The high value of hydroxyproline (149) might be indicative of hydroxyprolinemia. In this case, however, I find it more likely that Shawn's system is breaking down collagen and utilizing collagen residues to an abnormally high degree. If this condition isn't reduced after protein digestion and amino acid uptake are improved, then hydroxyprolinemia becomes a more convincing diagnosis. (Ref. Stanbury, Wyngaarden and Fredrickson, op.cit., 4th ed., Chapt. 17, pp 356-361.)

One important maladaptive protein food reaction that occurs with incomplete uptake of essential amino acids is due to unbalanced input to the citric acid cycle. Essential amino acids converge into this cycle via oxidative degeneration (catabolism). The citric acid cycle is the energy-exchange sequence where oxidation to produce CO_2 is coupled to phosphorylation to produce ATP.

An example of imbalanced citric acid cycle input is when the citric acid cycle is made deficient by lack of methylmalonyl-CoA (from methionine, valine, threonine, and isoleucine) or fumaric acid (from phenylalanine) or succinic semialdehyde (from glutamic acid and histidine), but when acetyl-CoA (from tryptophan, lysine and threonine) is present in normal amounts. In this example, the excess (unutilized) acetyl-CoA will tend to form ketones in the liver.

Probable Diagnoses and Suggested Treatments

Incomplete proteolysis (or absorption) of essential amino acids--

Supplemental digestive enzymes--stomach: pepsin (pepsinogen); pancreas: proteases such as chymotrypsin, trypsin (trypsinogen), also enterokinase and carboxypeptidases; intestine: peptidases.

Rotation diet: No special protein restriction yet evident; enhance zinc; enhance B_6, thiamine, niacinamide, vitamin C. Light

on yellow vegetables and leafy green vegetables; thyroid hormones may help the beta-carotene to vitamin A blockage.

Amino acid supplements suggested: Moderate amounts of lysine, leucine, isoleucine, valine, tryptophan, phenylalanine, glutamic acid, and proline, with 3 gms/day of citric acid.

Small amounts (tested first) of histidine, threonine, arginine and cystine (instead of methionine). Test dose should not exceed 0.5 gm; NH_3 could be a problem.

Do not feed the severe ammonia dumpers: glutamine, asparagine, glycine and serine.

After Shawn's digestion improves, take another 24 hour urine collection and have the quantitative analysis done. Then, it can be studied for enzymatic problems.

Jon B. Fangborn Ph.D.
Jon B. Fangborn

Distribution:
Dr. Philpott: 2 cys
original: JBP

CASE IV

PHILPOTT FOUNDATION
PHILPOTT MEDICAL CENTER
William H. Philpott, M.D., Director
820 N.E. 63rd Street
Oklahoma City, Ok. 73105
405 / 840-4357

RODERICK L.
4-22-81

INITIAL EXAMINATION AND HISTORY

Roderick L. is a two year old white boy examined on 4-22-81. His father and mother and Roderick were present during the interview. The chief complaint is that of diarrhea and intestinal bleeding in response to several foods and medications. The mother reports that six months prior to her gestation with him she had been on birth control pills for a period for three months, and she quit their use because she had edema of the abdomen. During gestation, she had nausea, headache and an infection on her foot that required one month to heal which was unusual for her. She usually healed well. There was a ten month's gestation period. He weighed 7 pounds, 11 ounces and was considered normal at birth. He was breast fed for two weeks only then placed on Similac for six months, then a trial given on Advance, which gave no change. Nursoy was given a trial at the age of 13 months. On the trial on Nursoy he vomited and had red specks in the stool. At present, his stools are still loose but not diarrhea unless he reacts to a food. His behavior has been that of hyperactivity since birth. He is much better on the present foods and can now listen and learn. I noted at times when his behavior was at its worse he also would fall easily, giving evidence of a motor involvement in the disorder. Medical attempts have been first of all Dr. Thomas Thurston, M.D., Pediatrician of Norman, Okla who cared for him for one year. He treated him for a diaper rash and made at least one attempt at changing the formula which achieved no value. He was taken to Mercy Hospital with an acute abdomen and diarrhea at one time. Then, for a period of three weeks was under the care of Charles Freeman, M.D., Pediatrician of Oklahoma City. He ran cultures of the stool and the ears and found no significant problem. The ears were swollen and he was referred for tubes in his ears. For five months, he was under the care of Mira Bhatia, M.D., Gastrointerologist of Oklahoma City. He was tested for parasites, complete GI studies and biopsy of the small intestine. No specific diagnosis was given. He was placed by her on sucrose, cow's milk and cereals. He continued to be hyperactive, even though the stools did improve. When the tubes were placed in his ears, no infection was found, only swelling and redness. When the antibiotic, Pediazole, was tried, he developed a fever of 104 and a rash. At the age of 17 months he was to William Rae, referred by Howard Hagglund, M.D. At that time he was vomiting and had diarrhea. Skin tests were used with neutralizations. He was found to be reactive to a large number of items. There appeared to be no appreciable benefit from this treatment. At 18 months, he was examined by iridology by a nutritionist, M. Haeworth of Tulsa. They were told that he likely had Crohn's disease, and a diet was provided. Considerable help has been achieved by this diet and he now is not very often hyperactive, and his stools

although loose are not the quality of a diarrhea. Among his habits is that he drinks a lot of water. It has been noted that foods that are freshly prepared are less potential reactive than foods that have been prepared and canned. Some of his known reactions are that of cereals producing a diarrhea and intestinal bleeding. Cow's milk producing hyperactivity, diarrhea and sleepiness. Goat's milk producing hyperactivity. Citric fruits produce a diarrhea and bleeding. Bananas produce a diarrhea. Chicken is questionably positive. It was positive on the skin but not observably positive when fed as a food. He uses a multiple vitamin called Solgan which contains no sugar or starch. He has ½ teaspoon of this powder two times a day. Dolomite, ½ teaspoon of the powder two times a day. He receives acidophilus liquid (Radiance) one teaspoon three times a day. From inheritance standpoint, there is no diabetes. A maternal great-aunt has colitis. His father has a diarrhea from onions.

RODERICK
PRIMARY DIAGNOSIS
June 8, 1981

I.

535.4 Gastritis - allergic

II.

555.0 Crohn's disease of duodenum and ileum

III.

526. Colitis, ulcerative

IV.

579.0 Celiac Disease
There was a drop of 79 points in alkaline phosphatase after feeding of wheat.

V.

790.2 Chemical Diabetes Mellitus - stress type
Results of deliberate food testing of single food meals.
mg% blood sugar one hour after each meal:

plums - 170 mg%
bananas - 185 mg%
maple sugar - 170 mg%
cane sugar - 160 mg%
okra - 160 mg%
mature corn - 195 mg%
rye - 160 mg%

VI.

271.3 Lactase Deficiency
Blood sugar mg% only reached 109.
Should be 120 mg% or above.

VII.

995.3 Allergy unspecified
Ingested food reactions other than dermatitis:
plums - crying
peaches - cried and fussed, was irritable
bananas - was irritable
orange juice - stomach hurting, it was large and bloated
applesauce & apple juice - stomach hurting
blueberries - more active, very lively
grapefruit - crying, sobbing, wants to be held constantly, staring
cantaloupe - sore abdomen, crying a lot
strawberries - nose running, crying, cranky, impatient, walking unstable

995.3 Allergy unspecified (cont.)
Ingested food reactions other than dermatitis:
potatoes - more active
okra - became real quiet, went to sleep shortly after eating, blood sugar was 160 mg%
mustard greens - irritable, crying, some yelling, doesn't know what he wants, wants to be held
acorn squash - sneezed and nose running
spinach - crying, irritable, runny nose
cashew butter - irritable, angry, throws things, cried for one hour, sweating, gas
triticale - crying, did not digest the triticale
oatmeal - irritable, will not mind good
wheat - more active, short attention span
rye - crying, irritable, blood sugar was 160 mg%
soy milk - possible blood in stool, doesn't want to play, sits still staring, not very alert, had instant bowel movement, went to sleep
milk - stomach hurts, hands are cold, blue around the mouth
cottage cheese - complained of stomach hurting
maple sugar - crying, screaming, runny nose, blood sugar was 170 mg%
cane sugar - crying, irritable, blood sugar was 160 mg%
honey - delayed reaction crying for one hour
fructose - thirsty a lot, runny nose

Cytotoxic - graded 0 to 4
3 - navy beans
2+ - chocolate, coffee
2 - cinnamon, apricot, beef, turkey
1+ - chlorine, cherry, lettuce, pinto beans, cottage cheese, milk
1 - Roquefort cheese, orange, apple, black walnuts, cabbage, beets, corn, oats, rice, barley, floride
Trace - goat's milk, tobacco, English walnuts, pecans, strawberry, grape mix, codfish, sole, mackeral, soybeans, peanuts, lima beans, potato, onion, wheat, mushrooms

Intradermals - showed positive on
TOE, Aspergillus, mold mix, Candida albicans

Sublinguals - positive reactions on
autohydrocarbons - laughing loudly, throwing toys, hyper

SECONDARY DIAGNOSIS

I.

267. Ascorbic acid deficiency was shown in urine analysis - 0 mg/dl. Normal is 40.

II.

269.3 Mineral Deficiency - results of hair analysis

	Results	Normals
potassium	5 mg%	6 - 23
zinc	7 mg%	13 - 20
iron	1.3 mg%	1.5 - 5
manganese	.03 mg%	.05 - .17
selenium	.03 mg%	.04 - .12
calcium	59 mg%	25 - 57
magnesium	11 mg%	3 - 7

III.

270.9 Amino Acids by column chromatography (plasma)
All essential amino acids are within reference range.

Other amino acids	Results	Reference range
cystine	2 MCM/100ml	3.1 - 14
phosphoethanolamine	absent	trace - 0.3
phosphoserine	1 MCM/100ml	0
hydroxyproline	1 MCM/100ml	0
cystathionine	trace	0
hydroxylysine	trace	0
1-methylhistidine	trace	0
3-methylhistidine	trace	0

Results of chemistry test

	Results	Reference range
alkaline phosphatase	211 units	35 - 148
SGOT	66 units	13 - 55
(indicates some liver dysfunction)		
CO_2 content	22 MMOL/L	24 - 34
creatinine	0.5 MG/DL	0.9 - 1.4 (males)
blood histamine	11 MCG/100ml	3.0 - 9.0

Hematology results

	Results	Normals
RBC	3.48	4.6 - 6.2
HCT	30.2	42 - 54
HGB	11.0	14 - 18

Results of reactive food on Heidelberg machine -
shows the food mature corn stayed in stomach 5½ hours.
Attempt to readjust pH caused vomiting.

Results of nonreactive food -
shows applesauce dumped as soon as eaten.

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820 N.E. 63rd Street
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Roderick

6-14-81

FORMULATION

He has been hyperactive since birth. He has had massive generalized gastrointestional reactions to cow's milk as well as soy. Provocative testing revealed abundant evidence of marked gastrointestional and behavioral reactions to a wide assortment of foods. He has a demonstrated celiac disease with a drop of 79 points in alkaline phosphatase when given wheat. A chemical diabetes mellitus stage was demonstrated by several foods producing a blood sugar of 160 mgs percent and beyond. Lactase deficiency was also demonstrated. Hair test biopsey reveals several minerals to be deficient. The serum tests for minerals were all normal. Surprisingly the test for vitamins was normal. One stress test for vitamin C following a food reaction revealed no vitamin C storage in the urine, revealing that he does dip into vitamin C deficienies under stress loads. The liver function test, both alkaline phosphatase and SGOT were abnormal. The liver disorder is likely secondary to this allergic type reactions to foods.

RECOMMENDATIONS

His foods should be rotated. Cereal grains and dairy products are to be left out of his diet. It may be that he may never be able to return to cereal grains. If and when milk is tried it should always be predigested with lactase. It should not be tried for some time. Subcutaneous or sublingual doses optimum doses for immunizations should be given on a once a week basis on candida albicans, mold mix, aspergillis and TOE. Ascorbic acid will be supplied as 2000 mgs. three times a day, B-6, B-2, niacinamide will be supplied. As minerals, potassium, zinc, manganese and selenium will be supplied. It is important that he receive proteases a head of a meal, this can be either bromaline, aloe vera or organic honey. Cystine will be an important supplement since he is deficient in cystine. Pancreatic digestive enzymes will be supplied. Acidyophois bacteria should be taken with each meal. He should be seen at frequent intervals and his case reviewed in depth in six months.

CASE V

PHILPOTT FOUNDATION
PHILPOTT MEDICAL CENTER
William H. Philpott, M.D., Director
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STEVEN S.

5-18-81

INITIAL EXAMINATION AND HISTORY

Steven is a twelve year old white boy examined on 5-18-81. His mother was present also during the examination. He is described as having been hyperactive all his life and having evidence of a learning disorder when he entered school. He has been under medical treatment for the hyperkinesis, first being tried on dextroamphetamines which caused a loss of appetite, then placed on Cylert which he has been on since 1976. The mother describes that during gestation, she had nausea, spotting and a bladder infection for which she was treated with sulfa. She had headaches daily. She was in labor twelve hours, and the labor was induced. This was her first child. At birth, it was noted that his chest was not fully developed. He would shake when he cried. He had to be fed hourly day and night. Characteristically, he was crying, upset, hyperactive and having a violent temper when he wanted to eat. He has been described as being hyperalert since birth, rocking, screaming, hyperactive, never satisfied, always busy as well as dependent. He was observed to be unduly frightened by faces. He was placed on a cow's milk formula which he spit up. Soy was tolerated. Cereals were introduced at two weeks, because he seemed so dissatisfied with his foods. Between ages one and two, he had a fungus infection on the skin. Tubes were placed in his ears as a small child. His adenoids were removed, and between the ages of two to five, he received frequent antibiotics for upper respiratory infections. An inguinal hernia was repaired at age two. His eyes do not focus together. There is a question of a possible grand mal seizure or other central nervous system type response occurring in 1976, following which he was noted to be sleepy and non-responsive to stimuli. He has remained hostile, dependent, demanding, sometimes throwing things. Educational type psychological examination in February, 1980 is summarized as follows: "This child has serious problems in auditory-verbal language processing. He cannot rapidly and clearly take in information, and subsequently to comprehend it. Memory is difficult and confused. Consequently, concepts are fragmented. His expressive language directly reflects this. Without an adequate oral language base, his success in school is limited, and he shows serious problems in reading and math. Compounding his language are specific difficulties with visual-motor integration, although compared to his verbal skills, this area is one of relative strength". From inheritance standpoint, his maternal great-aunt is diabetic. A maternal uncle was diagnosed as schizophrenic. A maternal great-grandmother was depressed in her old age. His father is described as having nasal allergies, chest pain, cardiac arrhythmias, urinary disorder and lactase intolerance.

My observations today are that he is quite overactive, oriented, narcissistically demanding. He has a normal gait.

STEVE (Age 12)

DSM III MULTIAXIAL EVALUATION

Axis I

134.01 Attention Deficit disorder with Hyperactivity
Hyperkinesis
Has been described as being hyperactive never satisfied, moving, rocking, screaming, since birth.

315.50 Mixed Specific Developmental Disorder
Delayed language acquisition
Difficulty in comprehending or in expressing verbal language. Below age level in reading
Below age level in arithmetic

292.84 Food Maladaptive reactions - Affective disorder
Apricots - evoked crying - grouchiness - upset.
Watermelon - withdrawn
Rice and Spinach - evoked uncooperativeness - grumpy.
Carrots - evoked impudent, silly reaction. Became progressively more obnoxious
Milk, chicken and broccoli evoked sadness and weeping.
Fresh corn and Pistachios - evoked tempermental attitude, frustration, and anger - was very difficult and uncooperative.
Maple sugar - hyper, ornery, uncooperative
Black olives - hyper and talkative.

Axis II

301.81 Narcissistic
Self-centeredness - demanding

790.2 Chemical Diabetes Mellitus Stress Type
Results of deliberate food testing of single food meals mg% Blood sugar 1 hr after each test meal.

A,

Foods	Results	Foods	Results
Cherries	180 mg%	Apricots	240 mg%
Pineapple	250 mg%	Dates	240 mg%
Spinach	160 mg%	Potato	180 mg%
Carrots	180 mg%	F. Corn	165 mg%
Rice	230 mg%	M. Corn	300 mg%
Maple sugar	163 mg%	Cane sugar	165 mg%

790.2 (Con't)

B. Ketones in urine* 3+ after eating corn 2+ after rice. (*3 hr. post meal)

C. Small intestine pH was 6; Normal 7.5 - 9
Low Bicarbonate production and therefore presumed low enzyme production.
This testing is consistent with chemical diabetes.

277.9 Unspecified disorder of metabolism
Errors of the metabolic process as manifested in reaction to:

A. Foods — Their reactions

Watermelon - sleepy, very clamy feeling
Carrots - sleepy and lethargic
milk - burping seeing doubles, fever

B. Cytotoxic graded 0 - 4
1 - coffee
trace - Tobacco, mackeral, navy beans, tomato, cabbage, cinnamon, clover honey, chlorine and floride.

784.0 Headache -
Turkey evoked pain in the head.

790.5 Other nonspecific abnormal serum enzyme levels
5/19/81 Alkaline Phosphatase was 241 (high) reference range is 35 - 148
5/20/81 Alkaline Phosphatase it was higher 277 - this was a rise of 36 points.

790.4 Nonspecified elevation of levels of transaminase
The SGOT was 122 units reference range 13-55
The SGPT was 141 units reference range 12-53

477.8 Other allergies - IgE mediated
RAST shows positive on Bermuda grass, and common ragweed.

796.4 Other abnormal serum findings.
Blood chemistry shows

CO_2 content (low)	23mmol/l	24 - 34
Uric acid	3.8mg/dl	4.0 - 8.5
Creatinine	0.8 mg/dl	0.9 - 1.4

HCT after meal of mature corn BS 300 38.9 Normal 42 - 54
WBC after meal of mature corn BS 3.0 Normal 4.0 - 11.6

269.2 Vitamin Deficiencies
B6 utilization disorder results of 24 hr urine shows Kynurenic Acid to 8,8 mg/24hr and Xanthurenic acid to be 4.0 mg/24hr reference range up to 3.
Vitamin C Ascorbic acid in urine 0 - 10 mg/dl normal 40
Serum Folic Acid was low 4.8 normal 5.0 - 24.0

269.3 Minerals - deficiencies - results of hair analysis

Test	Results	Normals
Low Calcium	18	25 - 57
Low Iron	1.3	1.5 - 5
Low Manganese	.03	.05 - .17
Low Chromium	.03	.04 - .07
High Sodium	36	12 - 35

270.9 Amino Acids - deficiencies and disorders
Serum Tryptophan low normal 1.4 mg/100ml reference range 1.4 - 3.0
Amino acids - results of 24 hr urine collected 5/21/81
Stressors - Frequently eaten foods plus 5 gm Tryptophan, 5 gm Histidine, and 1½ gm Methionine.

Essential Amino Acids	Results	R.R.
Isoleucine	14 mcm/24hr	18 - 210
Leucine	18 mcm/24hr	21 - 200

270.9(Essential Amino Acids Con't)

Below Reference Range	Results	Reference range
Methionine Sulfoxide	Absent	150 - 650
Asparagine	54 mcm/24hr	270 - 700
Glutamic Acid	11 mcm/24hr	55 - 270
Cystathionine	11 mcm/24hr	16 - 63
B-Alanine	Absent	11 - 70
Hydroxylysine	6 mcm/24hr	10 - 130
1-Methylhistidine	15 mcm/24hr	130 - 930
Above Reference Range	**Results**	**Reference range**
High Hydroxyproline	Trace	0
High Homocystine	Trace	0
High Gamma-Amino Butyric Acid	8 mcm/24hr	0

STEPHEN S.

LABORATORY DIAGNOSTIC METHOD

There were two days of stress by eating foods he commonly used with the second day adding stressors of methionine, tryptophan and histidine for the study of B6 utilization disorder and folic acid disorder, as well as discovering possible disordered amino acid function. After five days of eating infrequent foods, he was then given more frequent foods for provocative testing using single foods for each test with the blood sugar monitored one hour after each test meal.

FINAL FORMULATION

He has been ill since birth having been observed as being hyperactive and hyperalert. He has been prone to excessive motor sterotype movements. Upper respiratory infections have been a part of his illness. It seems obvious that he was reactive to cow's milk from the very first and reacted to cereal grains when they were introduced.

Provocative exposure to foods and chemicals revealed a cause and effect relationship between his major symptoms and maladaptive reactions to an assortment of environmental substances. Hyperkinesis was evoked by several foods, as was depression and negativism. A large number of foods evoked hyperglycemic reactions even as high as 300 mg% at one hour post-meal. Deficiencies or utilization disorders of B6, folic acid and vitamin C were in evidence. Hair test biopsy revealed deficiencies in calcium, iron, manganese and chromium. Amino acid profile revealed deficiencies in leucine, isoleucine, low glutamic acid and a trace of homocystine.

FINAL RECOMMENDATIONS

He should be supplemented with vitamin C, B6, folic acid, calcium, magnesium, copper, zinc, iron and chromium. Tryptophan at bedtime will likely improve sleep. A protease given ahead of a meal will aid in reducing reactions to foods. Cystine should be supplemented in view of low cystathionine. Improved digestion of proteins will be aided by supplemental whole amino acids. Protection against reactions can be provided by sublingual Heparin when exposure to reactive substances is anticipated. Thyroid will likely raise the temperature and increase his energy.

Name: Steve

Jon B. Pangborn, Ph.D.
August 24, 1981

Laboratory Data: Quantitative 24-hour urine amino acids, 5-21-81, loaded (tryptophan, histidine, methionine), Bio-Science Laboratories; other laboratory data from Philpott Medical Center

Referring Physician: William H. Philpott, M.D., Philpott Medical Center, Oklahoma City, OK

Analytical Observations

The urine amino acid analysis of this patient shows patterns characterizing somewhat incomplete digestive proteolysis and suggesting problems in amine transfer to form glutamic acid. The thyroid function is suspect due to deficient phenylalanine and iron (per the hair mineral analysis). There are also signs suggesting inadequate methylation or weak transmethylase enzymes. The metabolism of methionine at the homocysteine to cystathionine step is incomplete.

Observed are the following:

1) With respect to incomplete proteolysis, the following essential amino acids are deficient: leucine (18), isoleucine (14), and phenylalanine (24).

2) With respect to amine transfer, glutamic acid (11) is deficient.

3) With respect to inadequate methylation: 1-methylhistidine (15) is deficient although histidine (745) is within normal limits; creatinine (0.8 mg/dl) is low although arginine (6) and glycine (684) are within normal limits.

4) With respect to the metabolism of methionine, cystathionine (13) is low and homocystine (trace) is present.

Discussion

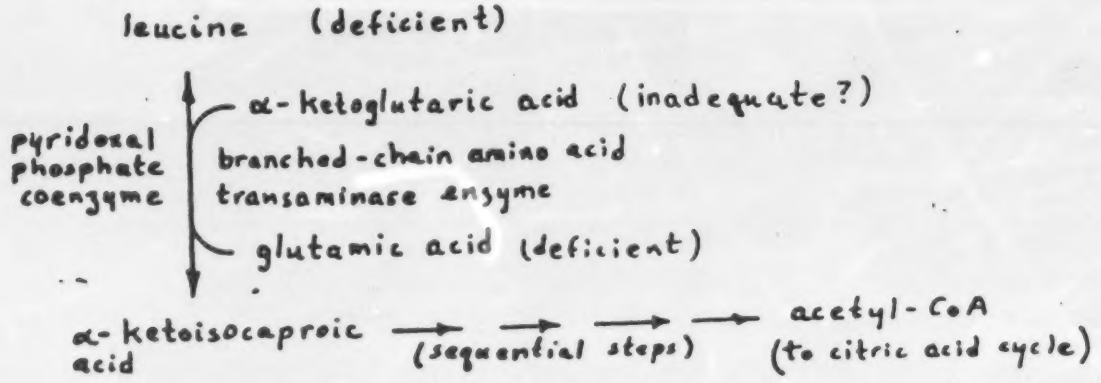
The Examination and History of this patient discusses definite and severe symptoms of uncontrollable behavior, hostility, and learning disabilities. Sometimes, amino acid analyses can be interpreted to find definite indications of metabolic errors or deficiencies that relate to the illness. In this case, however, there are no such definite evidences, except possibly the very deficient glutamic acid. There are other clues and mild metabolism deficiencies indicated by the data.

Incomplete proteolysis during digestion is probable per the deficiency in three (of ten) essential amino acids: leucine, isoleucine, and phenylalanine. The methionine value (33) seems low considering that an oral loading of this amino acid was administered. When methionine and isoleucine are deficient, their common metabolite, methylmalonyl-CoA, is likely to be deficient in supplying the citric acid cycle with succinyl-CoA. When leucine and phenylalanine are deficient, their common metabolite, acetyl-CoA, is likely to be deficient in supplying the citric acid cycle with a component needed for citric acid synthesis.

A result of the deficient essential amino acids could be a limitation in the functions of the citric acid cycle. Alpha-ketoglutaric acid, needed for amine transfer with coenzyme pyridoxal phosphate, may not be produced in sufficient amount; following from this, glutamic acid would be (and is) deficient; and chemical respiration and oxidative phosphorylation to generate CO_2 and ATP could be deficient.

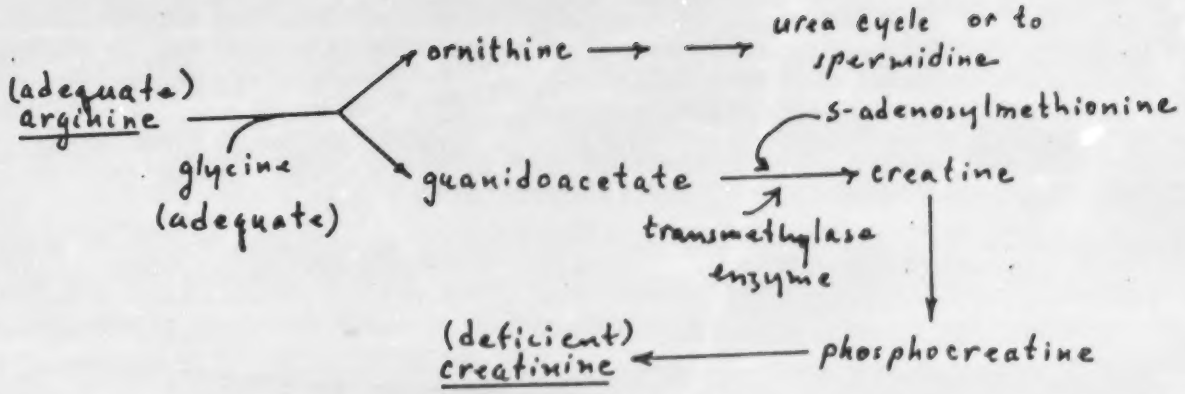
Complete proteolysis of food protein requires the secretion of pancreatic enzymes into the duodenum and the secretion of peptidases in intestinal mucosa. To be operative, these enzymes must be exposed to the correct pH (not to acidic or basic), and most are activated by zinc, in which this patient's hair shows to be low-normal. Others are activated by manganese, which is deficient per the hair mineral analysis.

Below, as an example of amine transfer, is the first chemical step that should occur in the catabolism of leucine. To form α-ketoisocaproic acid, leucine needs its transaminase enzyme, its B_6 coenzyme, and α-ketoglutaric acid (from the citric acid cycle).



If leucine (and B_6) are supplemented, the catabolism would still not proceed without α-ketoglutaric acid. Without α-ketoglutaric acid, glutamic acid will not be produced. In the brain, the major pathway for ammonia detoxification is formation of glutamine from glutamic acid. With low glutamic acid the patient is subject to (protein-induced) episodic hyperammonemia. This may be occurring although symptoms would not necessarily be prompt or discernable after an NH_3 or protein challenge. Considering the diet test foods of the urine collection day (5-21-81), which were: bananas (NR), blueberries (NR), potatoes (++), and garbanzos (NR); there probably was not enough protein to provide a protein challenge.

Methylation of amino acids is needed in several metabolism sequences: remethylation of homocysteine to methionine, synthesis of choline from ethanolamine, synthesis of creatine from guanidoacetate, and methylation of histidine are examples. The amino acid analysis indicates normal ethanolamine. Homocystine is present as trace. This could be a B_6 utilization problem or it could be a weak sign that homocystine is not being adequately remethylated. In the latter case, however, one would expect cystathionine to not be low (as the data show it). Hence, a mild problem in the utilization of B_6 in converting homocystine to cystathionine is more likely. In the case of low creatinine and normal arginine and glycine, however, methylation (requiring s-adenosylmethionine and



a transmethylase enzyme) is likely at fault. A coenzyme would be tetrahydrofolate (folic acid); folic acid is noted as deficient per the blood vitamin analysis. Also needing s-adenosylmethionine, a transmethylase enzyme, and probably folic acid, is the methylation of histidine to 1-methylhistidine; 1-methylhistidine is below normal limits while histidine is normal.

A final observation is that asparagine is deficient even though its precursors, glutamine and aspartic acid, are normal. Magnesium, the enzyme activator for asparagine synthesis shows as normal in the hair analysis. Hence, the reason for low asparagine is not evident.

Probable Diagnoses and Suggested Supplements

Definite

Deficient glutamic acid formation possibly resulting from deficient α-ketoglutaric acid, lack of amine transfer, or nonutilization of vitamin B_6 as coenzyme pyridoxal phosphate, and possibly resulting in brain ammonia intoxication.

Probable

1) Incomplete digestive proteolysis possibly related to deficient manganese as a digestive enzyme activator.

2) Limited methylation capacity in histidine metabolism, possibly also in synthesis of creatinine.

3) Limited or disordered thyroid function with respect to iodated tyrosine residues (T-1,2,3,4 hormones) due to low phenylalanine and deficient iron.

Possible

Utilization problem for pyridoxal phosphate in the methionine metabolism pathway at homocysteine to cystathionine.

Supplements

Amino acids: Test singly 2 g of glutamic acid; if no reaction is evoked, then 0.5 g at each meal. Test singly 2 g of asparagine; if no reaction is evoked, then 0.5 g at each meal. 1 g/day leucine, isoleucine, phenylalanine.

Citric acid cycle: 3 g/day of citrate salt or citric acid.

Vitamins: B_6, folic acid, niacin, C.

Minerals: Fe, Mn, Cr, Zn, Cu.

Distribution
2 cys: Dr. Philpott
orig: Dr. Pangborn

S.J

TOXIC METHYLATED AMINES